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PAROXYSMAL PULMONARY ŒDEMA AND ITS TREATMENT. By ALFRED STENGEL, M.D. 1

THE CONTROL AND TREATMENT OF HYPERTENSION AND ARTERIOSCLEROSIS. By HENRY L. ELSNER, M.D. 22

THE TONIC USE OF DIGITALIS. By EMILE SCHMOLL, M.D. 35

THE TREATMENT OF TYPHOID FEVER. By FRANK SHERMAN MEARA, M.D. . . . 44

CHOLERA FROM A MODERN STANDPOINT. By ALVAH H. DOTY, M.D. 67

THE RELATION OF DISEASE OF THE GALL-BLADDER AND BILIARY DUCTS TO THE GASTRIC FUNCTIONS. By JOHN A. LICHTY, M.D. 72

THEI DYSPEPSIA OF OLD AGE. By GEO. M. NILES, M.D. 79

THE DIAGNOSIS OF TUBERCULOSIS OF THE BRONCHIAL GLANDS. By HENRY FARNUM STOLL, M.D. 83

PELLAGRA, WITH A REPORT OF TWO CASES WITH NECROPSY. By PAUL V. ANDERSON, M.D., and WILLIAM G. SPILLER, M.D. 94

THE INFLUENCE OF THE TRENDELENBURG POSITION ON THE QUANTITY OF URINE EXCRETED DURING ANESTHESIA. By J. WESLEY BOVÉE, M.D. 106

MULTIPLE ABSCESSES OF THE NASAL SUBMUCOSA IN A CASE OF LEUKEMIA. By JOSEPH P. TUNIS, A.B., M.D. 109

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BY EMINENT AMERICAN AND ENGLISH AUTHORITIES

EDITED BY

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Professor of Therapeutics and Materia Medica, Jefferson Medical College, Philadelphia; Physician to the Jefferson Hospital; Author of A Text-book of Practical Therapeutics, A Text-book of the Practice of Medicine, etc.

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BRIEF OF CONTENTS

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CONTENTS.

ORIGINAL ARTICLES.

Paroxysmal Pulmonary Œdema and its Treatment 1
By Alfred Stengel, M.D., Professor of Clinical Medicine in the University of Pennsylvania, Philadelphia.

The Control and Treatment of Hypertension and Arteriosclerosis . 22
By Henry L. Elsner, M.D., Professor of Medicine in the Syracuse University; Physician to St. Joseph's Hospital; Consulting Physician to Women's and Children's Hospital, Syracuse, N.Y.

The Tonic Use of Digitalis 35
By Emile Schmoll, M.D., Assistant Professor of Medicine in the Cooper Medical College; Physician to the City and County Hospital, San Francisco.

The Treatment of Typhoid Fever 44
By Frank Sherman Meara, M.D., Professor of Therapeutics in the Cornell University Medical College, New York.

Cholera from a Modern Standpoint 67
By Alvah H. Doty, M.D., Health Officer of the Port of New York.

The Relation of Disease of the Gall-bladder and Biliary Ducts to the Gastric Functions 72
By John A. Lichty, M.D., Associate Professor of Medicine in the University of Pittsburg.

The Dyspepsia of Old Age 79
By Geo. M. Niles, M.D., Professor of Physiology in the Southern College of Pharmacy; Associate Professor of Physiology in the Atlanta School of Medicine; and Gastro-enterologist to the Tabernacle Infirmary, Atlanta, Georgia.

The Diagnosis of Tuberculosis of the Bronchial Glands 83
By Henry Farnum Stoll, M.D., Assistant Physician to the Hartford Hospital; Physician to the Tuberculosis Clinic, Hartford Dispensary, Hartford, Conn.

Pellagra, with a Report of Two Cases with Necropsy 94
By Paul V. Anderson, M.D., of Morganton, N. C., and William G. Spiller, M.D., of Philadelphia.

The Influence of the Trendelenburg Position on the Quantity of Urine Excreted During Anesthesia 106
By J. Wesley Bovée, M.D., Professor of Gynecology in the George Washington University, Washington, D. C.

Multiple Abscesses of the Nasal Submucosa in a Case of Leukemia . 109
By Joseph P. Tunis, A.B., M.D., Fellow of the College of Physicians of Philadelphia.

REVIEWS.

Preparatory and After-treatment in Operative Cases. By Herman A. Haubold, M.D. 114
The Prescribing of Spectacles. By Archibald Stanley Percival, M.A., M.B., B.C. 116
A Treatise on Diseases of the Eye. By John Elmer Weeks, M.D. 116
The Prevention and Treatment of Abortion. By Frederick J. Taussig, A.B., M.D. 117
Spondylotherapy. Spinal Concussion and the Application of Other Methods to the Spine in the Treatment of Disease. By Albert Abrams, A.M., M.D. 118
Grundriss und Atlas der Speciellen Chirurgie. By Professor Dr. George Sultan 120
Congenital Dislocation of the Hip. By J. Jackson Clark, M.B. Lond., F.R.C.S. 120
The Psychology of Dementia Præcox. By Dr. C. G. Jung 121
De l'Usage en Clinique de l'Ultra-microscope en Particulier pour la Recherche et l'Etude des Spirochetes. Par le Dr. J. Comandon . . . 122
The Open-air or Sanatorium Treatment of Pulmonary Tuberculosis. By F. Rufenacht Walters, M.D., B.S., M.R.C.P., F.R.C.S. 122
Refraction and Motility of the Eye, with Chapters on Color Blindness and the Field of Vision, Designed for Students and Practitioners. By Ellice M. Alger, M.D. 123
Surgical Anatomy. By John A. C. Macewen, B.Sc., M.B., C.M. 124
Mammalian Anatomy, with Special Reference to the Cat. By Alvin Davison, Ph.D. 125
L'Hypophyse et la Médication Hypophysaire. By Arthur Delille . . . 125
A Text-book of Pathology. By Joseph McFarland, M.D. 126
A Text-book upon the Pathogenic Bacteria. By Joseph McFarland . 126

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D., AND W. S. THAYER, M.D.

A New Sign of Inflammation of the Meninges 127
Chronic Antimony Poisoning in Typesetters 127
The Treatment of Malaria with "606" 128
A New Method of Administering "606" 128
The Treatment of Strychnine Poisoning by Insufflation and Curare . . 129
The Effect of Malignant Tumors upon the Blood 129
Malarial Pigment 130
Intestinal Amœbiasis 130
Test for Gastric Cancer 130
A Comparison of the Results Obtained with the Different Sphygmometers 131
The Influence of the Position of the Body upon Evacuation of the Stomach 132

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D., AND T. TURNER THOMAS, M.D.

Circular and Lateral Suture of a Vein 132
An Explanation of the Pathogenesis of Appendicitis Based upon Experimental and Bacteriological Investigations 133
Anuria after Gallstone Operations 134
The Mobile Cecum as a Cause of Many Cases of Chronic Appendicitis . . 135
Concerning the Value of a Simple Continuous Suture Passing through the Whole Vessel Wall 135
Cancer of the Prostate 136
Transthoracic Cardiotomy, a New Method for the Cure of an Impassable Cicatricial Stricture of the Œsophagus 137
Saccular Autoplasty in Necrosis of the Intestine from Strangulated Hernia 138

THERAPEUTICS.

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.

The Treatment of Syphilis with "606" 138
The Treatment of Syphilis with Ehrlich's "606" 139
The Subcutaneous Use of "606" 140
The Treatment of Sciatica with Epidural Injections 140
Chlorosis 140
The Present Status of Antityphoid Inoculation 141
The Influence of Age and Temperature on the Potency of Antidiphtheritic Serum and Antitoxin Globulin Solution 141
Deep Perineural Injections for the Relief of Neuralgia and Sciatica . . . 142
The Action of Cod-liver Oil in Rachitis and Spasmodic Affections of Children 142

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.

The Diagnosis of Nasal Diphtheria in the Newborn and in Infants . . 143
Immunized Human Serum in Hereditary Syphilis 143
The Diagnosis of Epidemic Meningitis in Infants 144
An Epidemic of Anterior Poliomyelitis in Austria 145

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.

Pernicious Nausea of Pregnancy Treated by Thyreoidin 146
Tetany of Pregnancy 146
Combined External and Vaginal Version 147
The Treatment of Fibroids Complicating Pregnancy 148

GYNECOLOGY.

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.

An Improved and Perfected Operation for the Relief of Extreme Cases of Procidentia, Cystocele, and Rectocele 149
The Radical Cure of Hemorrhoids: Modified Whitehead's Operation . . 150
The Wertheim-Schauta Operation 150
The Structure of the Stroma of the Endometrium and its Bearing on the Menstrual Changes 151

OPHTHALMOLOGY.

UNDER THE CHARGE OF

EDWARD JACKSON, A.M., M.D.,

AND

T. B. SCHNEIDEMAN, A.M., M.D.

Self-inflicted Ocular Traumatisms among Russian Conscripts 152
Pigmentation of the Retina in Nephritis 152
Isolated Monolateral Paralysis of the Third Nerve of Aneurysmal Origin 153
Detachment of the Retina Treated by Tuberculin 153
The Principal Meridians in Astigmatism in Relation to the Lateral Inclination of the Head 153
Unilateral Cataract from Electricity 154

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

WARFIELD T LONGCOPE, M.D.

The Interrelation of Glands of Internal Secretion. III. Chemotaxis . . 154
The Venous Pulse in Fibrillation of the Auricle in the Mammalian Heart, with Reference to the Venous Pulse in Pulsus Irregularis Perpetuus 155
The Keith-Flack Node in the Production of Cardiac Arrhythmia . . . 155
The Substitution of Strontium for Calcium in Bones 156

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ORIGINAL ARTICLES.

PAROXYSMAL PULMONARY ŒDEMA AND ITS TREATMENT.

By Alfred Stengel, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

The condition designated paroxysmal pulmonary œdema cannot well be mistaken for any other. It comes on very suddenly with little or no premonitory indication and rapidly assumes such alarming characters that the patient and those about him feel that a fatal termination can be a matter of but few minutes. The patient at the very beginning experiences a sense of suffocation or oppression of breathing which speedily becomes extreme. He struggles for breath, and soon becomes additionally oppressed by paroxysms of suppressed cough in which a little frothy and often blood-tinged serous fluid is brought up. Later, larger quantities of the same sort of frothy serum may be expectorated or may literally well up from the lungs. The breathing is merely moist on auscultation or noisy, according to the degree of œdema; in severe attacks it becomes suppressed. When the frothy expectoration has stood for a time in a vessel the bubbles of air disappear, leaving a clear and usually blood-tinged serous fluid.

At the moment of the attack the patient grows suddenly deathly pale and wears the anxious expression of terror of one who fears instant death. The hands, feet, and whole body grow cold and become bathed in a dripping sweat. Later, as the respirations grow more and more difficult, cyanosis becomes associated with the pallor. Extreme cyanosis, however, is uncommon. The pulse, usually extremely weak and rapid in the beginning, may continue so until some relief is afforded. In some of the seizures in cases reported in this series the pulse during the early part

of the paroxysm was almost imperceptible, and improved very gradually as the attack subsided. Occasionally, however, the pulse may be rapid and excited during an attack and may give no evidence of reduced cardiac power. It will be shown later that even in such cases the actual working power of the left ventricle may be diminished, and that the mechanical conditions capable of developing pulmonary œdema may be present despite the seeming strength of the pulse.

At the beginning of the attack the patient is usually nervous and frightened, and this condition may increase with the oppressed breathing and suffocating cough. Later, a certain amount of stupor or partial unconsciousness may develop, but this is unusual. After a time the patient grows easier; warmth returns to the hands, which have been cold and cyanosed; the breathing improves; the moist rales in the chest grow less abundant; color returns to the face; and the pulse grows stronger and less frequent. The majority of paroxysms terminate in this way; in but a small proportion of cases does a fatal termination occur, notwithstanding the extreme violence of the symptoms usually observed. Some patients have, however, died in the first attack; others have survived for a period of months or years, despite repeated seizures. In Lissaman's[1] case there were seventy-two attacks during a period of over two years; and the patient was living at the time of the report. In one of my cases paroxysms have occurred at intervals of weeks or months during the last ten years; and the patient is now in rather better physical condition than after the first attack in 1900.

The distinguishing features, clinically speaking, of the condition that has been referred to are its sudden onset, usually with slight provocation; the evidence of intense pulmonary œdema; the expectoration of quantities of frothy and blood-stained serum; and the repetition of such attacks without intercurrent complicating conditions. These features distinguish this condition from certain other forms of acute pulmonary œdema, which, during their height, may not be especially different in clinical manifestations.

The seizures, as a rule, come on in the evening or after the patient has gone to bed. He may awake from a profound sleep with an oppressed cough, followed by the symptoms described. In some cases, vague apprehensions or unaccountable nervous feelings may precede the attack; but these usually occur only after a repretition of the seizures has made the patient familiar with their character and fearful of their recurrence. Various kinds of excitement, physical or mental, may provoke the attacks; but in a large proportion of cases there is no such cause.

Repeated attacks of the same character may occur at intervals of days or weeks during a long period of time. In one of my cases

[1] Lancet, February 8, 1902.

the seizures began over ten years ago and have continued up to the present time at varying intervals. Between attacks the patient may be perfectly well, or more frequently give evidence of some inadequacy of cardiac compensation. In the cases which have terminated fatally the final result has usually been due to increasing cardiac decompensation rather than to the sudden effect of an attack of pulmonary œdema.

It is necessary to point out here some of the other varieties of acute pulmonary œdema met with clinically, so that the limitations of what is intended to be included under the title paroxysmal pulmonary œdema may be more evident.

1. Acute Œdema Associated with Other Lesions of the Lungs. It is a well-known fact that cases of pneumonia or pulmonary tuberculosis may terminate suddenly by the development of an acute œdema affecting a part or all of the lung not involved in the original trouble. The explanation offered for such cases by older writers, and to a large extent still accepted, is that there is a collateral hyperemia or fluxion, resulting from the extent of the original trouble. Another explanation is, however, possible, particularly in cases of acute pneumonia. This is that, as a result of dissemination of the infective cause, an acute inflammatory œdema of the entire lung or of both lungs follows after the original infection. In this condition, there may be a close resemblance in the symptoms to what has been described before; but the history of the case and the circumstances under which the acute œdema arises distinguish this from paroxysmal œdema.

2. Acute Infectious Œdema; Acute Inflammatory Œdema; or Acute Œdematous Inflammation. This condition, which was originally described by Traube as serous pneumonia, or diffuse congestive pneumonia, may occur in association with certain infectious diseases, such as scarlet fever, smallpox, typhus fever, rheumatism, or influenza, or as a primary infection of the lungs. Its resemblance to vagus pneumonia is too close to be overlooked; and its occurrence in cases of acute cerebral disease, head injuries, or sunstroke is likewise suggestive. Strümpell[2] alludes to such cases, and mentions the absence of other lesions than the œdematous lung. Hilton Fagge[3] and Fowler[4] describe the same condition. Sahli[5] insists that this is purely an infectious condition; and Kockel,[6] in his pathological and bacteriological study of 6 cases following head injury, found a constant association of bacterial invasion and the acute inflammatory lesions. He further states that Thoma believes that most diffuse or extensive œdemas are inflammatory and infectious, and mentions that Ziegler describes

[2] Text-book on Medicine, second American edition, p. 183.
[3] Text-book on Medicine, third edition, i, 1002.
[4] Diseases of the Lungs, p. 281.
[5] Archiv f. exper. Pharm. u. Path., 1885, 19.
[6] Deut. Naturf. und Aerzte, 1896, lxviii, 30.

such cases as "septic-toxic" œdema. Welch[7] states that he agrees with Sahli regarding the frequency of infectious inflammatory pulmonary œdemas, basing his opinion upon the systematic bacterial examinations made at all autopsies at the Johns Hopkins Hospital. Not only in irregular and localized œdemas, but also in not a few extensive and even general ones, plate cultures from the lungs showed numerous colonies of bacteria, most frequently streptococci and lanceolate micrococci, so numerous that they must have been in active growth in the lungs. Sudden œdema associated with Bright's disease, described by Dieulefoy[8] and by other French writers, may, in some cases, belong in this same group, but is possibly susceptible of another explanation, similar to that which will be suggested in the case of paroxysmal œdema.

The clinical manifestations of acute infectious œdema, as I have seen them in some cases of influenza and other infections, do not differ greatly from those of paroxysmal pulmonary œdema, except that there is much less suddenness and vehemence of the symptoms. Certain authors (as Romberg) have mentioned as distinguishing features the fact that there is fever in these cases, and usually a pulse suggestive of increased rather than lessened cardiac action; and undoubtedly these are important points, and may lead to a proper diagnosis. It must, however, be recalled that fever is not invariably present, and that in some instances of paroxysmal pulmonary œdema the pulse-rate does not give evidence of decided cardiac weakness. A more important criterion for distinguishing the two conditions is the tendency to recurrence in the one and the absence of such a tendency in the other. Furthermore, paroxysmal pulmonary œdema, as will be pointed out later, occurs in association with certain other conditions, cardiovascular or renal, and rarely independently of such causes; while infectious œdema may appear in persons previously in perfect health. Later, in the discussion of the pathogenesis of the pulmonary condition in paroxysmal cases, I shall have occasion to point out that infection has been thought of as the important etiological factor. A series of paroxysms would, therefore, necessarily imply a series of successive infections. Such an occurrence is manifestly most unlikely, and all the facts connected with the recurrent paroxysms in cases such as those observed by myself over long periods of time speak strongly against this possibility.

3. Acute Œdema Accompanying Rapid Dilatation of the Heart. In cases of acute dilatation of the heart caused by acute myocarditis or degeneration of the heart, considerable œdema of the lungs may develop quite rapidly; and the clinical features at the height of such an attack may be indistinguishable from those

[7] Quoted by Meltzer, "Œdema," American Medicine, viii, Nos. 1 to 5.
[8] Path. Int., thirteenth edition, T. I., i, 282.

which occur in seizures of paroxysmal œdema such as have been described. The manner of onset and the coincident conditions, however, distinguish such cases from the paroxysmal type; for there is usually a clear history of preceding acute cardiac weakness, and, perhaps, even prior to that, of some infection which has occasioned it. Then, too, physical signs of increasing cardiac weakness and of increasing dilatation of the heart precede the onset of the pulmonary œdema. Thus, it is usually possible to distinguish the two conditions, notwithstanding the fact that at the height of the symptoms they are practically indistinguishable, as cardiac dilatation quite commonly occurs as an incident of the attack in the paroxysmal variety.

While we may thus recognize four types of sudden, or acute, œdema of the lungs, it must be apparent that the first is clearly distinguished from the others mainly by its recurrent character; and for this reason, I have ventured to use the term *paroxysmal pulmonary œdema.* Among other names that have been suggested is acute suffocative pulmonary œdema, but this is less descriptive, because practically all forms of acute œdema are almost equally suffocative in character.

A report of some typical cases will serve to indicate the clinical features.

Case I.—Mr. E. B., aged seventy years, at the time of his death, in May, 1909, had been under my observation for six or eight years. He had formerly been addicted to the overuse of alcohol, and was an inveterate smoker. He came of a distinctly gouty family, and he himself suffered from a moderate obesity and later from a mild glycosuria which came and went. During the time he was under my observation I recognized the development of increasing arteriosclerosis, and examinations of the urine showed traces or small quantities of albumin and tube casts significant of an increasing interstitial nephritis. The blood pressure was constantly high, and increased with succeeding years. During the last four years of his life the sugar had disappeared from the urine. None was present from January, 1905, up to the time of his death.

The first attack of pulmonary œdema occurred during the winter of 1908, after a somewhat heavy dinner. The patient had gone to the theatre, and while there complained of the heat of the room and of oppression of breathing. He was quickly hurried out before the attack developed, but he could not get to his home, and when I reached him, fifteen minutes later, in an adjoining house, where he had been taken, I found him in the midst of an attack of the most intense severity. The pulse was scarcely palpable, the face was livid, and the skin everywhere icy cold and bedewed with dripping perspiration. The breathing was noisy and the lungs were evidently œdematous throughout. A physician who had

seen him before my arrival had given him a pearl of nitrite of amyl, which was inhaled from a handkerchief with apparently some slight relief. I gave him a hypodermic injection of morphine with atropine, and in a short time he was sufficiently restored to be taken home; but subsequent hypodermic injections of strychnine, nitroglycerin, and atropine were given, as the patient was greatly exhausted. After an hour he fell asleep, and the next day was practically well though much exhausted. After this attack the patient had a succession of similar seizures, probably fifteen in all, none, however, of the intensity of the first, and in each of these the prompt administration of hypodermic injections of morphine, sometimes with and sometimes without atropine, relieved him speedily. The general course of his case, however, was a downhill one, and finally increasing œdema and other evidences of cardiac failure terminated the condition. Toward the end it seemed to me as if he might perish in one of the attacks on account of his generally weakened condition and the progressive deterioration of cardiac strength, but there never was any difficulty in controlling the seizures themselves. The final outcome of the case and the progress was practically the same as that of a case of general arteriosclerosis with cardiac disease unattended with such paroxysms of pulmonary œdema.

Case II.—Mrs. H. E. R., now aged fifty years, first came under my observation in December, 1900. She had had a severe attack of cardiac disturbance of some sort a short time before that date, and when I first saw her she was still suffering from some of the consequences of a disturbance or a loss of compensation. The principal symptoms were dyspnœa, cough, and other indications of pulmonary congestion. On physical examination I found slight enlargement of the heart and the evidences of mitral stenosis. After the administration of moderate doses of cardiac tonics, compensation became restored and the patient was quite well until February, 1902, when a sudden attack of intense congestion and œdema of the lungs with cardiac weakness occurred in the middle of the night. Another attack occurred in March, 1902, after which she remained free of seizures, although at times not entirely well, until the spring of 1906, when she was greatly reduced by hemorrhoidal bleeding, and in May she had another severe attack of acute œdema. The fifth attack occurred during the summer of 1906, the seventh about six weeks later, the eighth and ninth in October and December of 1908, the tenth in October, 1909, and the eleventh on January 28, 1910. All of these attacks have been of similar character and all occurred during the night. In several the direct cause was sexual excitement. In others the patient has been wakened from sleep with a frightened feeling, which was quickly followed by intense oppression of breathing, a rattling sound in the chest, great weakness and rapidity

of the pulse, and the expectoration of more or less copious amounts of frothy and usually blood-tinged serum. Since the first attack, and up to the latest one, she has had the prompt attendance of a physician, who administered hypodermic injections of morphine with small amounts of atropine, and the result has invariably been a speedy recovery after a single hypodermic dose. In the last attack strychnine and nitroglycerin were used, and though the patient recovered from the attack, the duration was somewhat longer than on previous occasions, and the attack was one of no great severity, having been attended with but little œdema and no frothy expectoration. The first attack of all was described as the most severe of the series, but I cannot give any details regarding it. After the attacks the patient quickly regains her normal condition, but during the ten years she has been under my care there has been the constant evidence of a cardiac defect with slight dyspnœa on exertion and a lowered general vitality. There has been no evidence of progressive loss of power in the heart, and the patient's general condition now is apparently quite as satisfactory as it was in the beginning. The physical signs of mitral stenosis, as is so common in cases of that lesion, are more distinct in the intervals between than during or immediately following attacks. Urinary examinations have shown a slight trace of albumin on a few occasions, but not more than might be attributed to the leukocytes present as a result of some vesical trouble. The latest examination made on the day following her attack of January 28 showed: specific gravity 1018, reaction acid, no albumin, no sugar, no cylindroids or casts, a number of leukocytes, and some uric acid crystals. There has never been any indication in the urinary examinations or symptoms of nephritis.

Case III.—I. K., aged fifty years, first came under my care in 1901. He had used alcohol periodically to great excess, but not at all during the last year and never subsequently up to the time of his death in 1909. He had, however, continued smoking immoderately, and never limited this at all reasonably until near the end of his life. In 1902 he suffered from an attack of abdominal pain which Dr. J. Chalmers Da Costa and I regarded as due to appendicitis, but we decided it safer to avoid operation on account of certain cardiac symptoms. For some time previous to this date the patient had complained of substernal and precordial oppression, especially on slight exertion, as in walking up moderate hills on his golf course. The pulse was constantly rapid, and more so when he was specially oppressed. The heart sounds were weak but sharp, and the aortic second sound slightly accentuated. There was no cardiac enlargement and no murmur. In 1904 he had considerable anginoid pain, and in the summer another attack of what was regarded as appendicitis. Early in 1905 he had another abdominal attack, with pain most marked in the epigastrium.

Soon after this he complained of a severe headache or neuralgia, which was followed by right homonymous hemianopia affecting the right upper quadrant. Dr. de Schweinitz saw him and reported the condition "right lateral homonymous tetranopia." The vision improved after some time and possibly as a consequence of the use of potassium iodide. Another abdominal attack occurred later in 1905. In the meantime, his precordial distress never was wholly relieved, though it was less severe or he had grown accustomed to it. During the summer of 1907 he had one or two attacks of severe cardiac distress while at some altitude in the Adirondack mountains; and in the spring of 1908 he had the first attack of threatening character. The description of the seizure, given by a friend who was with him, corresponded in every detail with that which would apply to subsequent attacks which I observed. A pellet of morphine and one of nitroglycerin were given, and after some hours relief occurred. I saw the patient the next day and found him in his usual condition, though somewhat used up by his experience of the night before. The cardiac signs were the same as those described. On the following night another attack occurred, and in this he developed a complete right hemiplegia with aphasia. Up to this time I had not seen the patient in an attack, but during the following eighteen months I saw him in several, and in others Dr. Klaer was called.

There was usually little premonition of the attack, but a certain amount of nervousness and mild oppression preceded the paroxysm for a short time. The attack itself set in very suddenly with extreme oppression of breathing, violent palpitation with great weakness of the heart beat and pulse, ashy pallor combined with some cyanosis, and cold sweat. Very soon he would begin to cough in short interrupted paroxysms, bringing up frothy serous fluid of pink color, and in bad seizures the same kind of frothy fluid flowed from his mouth between the coughing attacks. At the height of the attack the lungs were filled throughout with moist rales and the entrance of air was reduced to a minimum. Scarcely any inspiration or expiration could be distinguished.

During the eighteen months following the hemiplegia there were twelve such attacks or distinct beginnings of attacks and a number of milder suggestions. He was under the constant care of a nurse, who had been instructed to give a hypodermic injection of morphine, $\frac{1}{4}$ grain, and atropine, $\frac{1}{250}$ grain, at the first sign of a seizure, to be followed by one of strychnine sulphate, $\frac{1}{20}$ grain, and nitroglycerin, $\frac{1}{1000}$ grain, within ten or fifteen minutes, and a repetition of morphine, $\frac{1}{8}$ grain, with atropine, $\frac{1}{250}$ grain, after a half hour if necessary. This plan never failed to abort the attacks within a few minutes and without any actual need for the second or third injection. In two or three of the attacks, when the nurse or members of the family thought the first symptoms merely

nervousness, the attacks got greater headway and relief did not occur as speedily, but even then the patient was relieved in a half hour. The effect of the injections was to cause a rapid improvement in the oppression of breathing, followed by a fuller pulse, returning warmth of the extremities, and diminution in the frothy expectoration. The last four attacks occurred during the summer in Atlantic City, and Dr. Porteous reports that these were of the same character as previous ones, and up to the last one were controlled by a moderately large hypodermic dose of morphine and atropine, which on one or two occasions had to be repeated. The last and fatal attack differed only in being more severe than any previous ones Dr. Porteous had seen. It began while the patient was in a theatre, and possibly the delay in applying treatment may have been responsible for the fatal result. There was no autopsy.

Case IV.—W. H. S. was admitted to my service at the University Hospital in February, 1905, on account of cough, expectoration, and occasional hemoptysis. The patient was a shipper, aged twenty-two years, who had suffered from articular rheumatism four years ago, had been a moderate user of alcohol and tobacco, but without any other significant history. At this time signs of aortic regurgitation and mitral stenosis were discovered and a suspicious lesion was found at the apex of the left lung. We were never certain that this was a tuberculous lesion, as examination for bacilli was always negative and the cardiac condition could have easily occasioned a localized area of congestion. The patient left the hospital in April and was treated at a sanatorium for tuberculosis, where he remained until September, 1905, having gained considerably in weight, and being then discharged as cured. He attempted to resume his work, but soon noticed a return of blood-tinged sputum, and reëntered the hospital in the fall of 1907, with practically the same conditions we had previously found. The patient, however, had increased greatly in weight from the forced feeding, and we were under the impression that this increased weight had exaggerated rather than helped his cardiac difficulties. After a month's stay in the hospital he felt quite well, and was about to go home, when some little excitement incident to his departure from the hospital caused a sudden attack of acute œdema. He had walked from the ward to the elevator, and there was seized with intense dyspnœa and weakness, and was carried back to his bed. A severe attack of acute œdema followed, the respirations increasing to 50, the pulse to 160 per minute. Considerable frothy material welled out of his mouth and he was extremely cyanosed. Repeated injections of camphorated oil, digitalin, and atropine were given. Of the last, doses of $\frac{1}{80}$ grain were given every thirty minutes during four hours, but the attack did not subside for eight hours, after which there was a slight improvement. During this attack it was noted that the cardiac dulness increased to the left

as well as to the right, and as the patient improved the following day this increased dulness seemed to diminish. A second attack occurred two weeks later, and was described as a very severe one. The symptoms were the same as those in the previous seizure. A hypodermic injection of morphine and atropine followed by digitalin and camphorated oil quickly terminated this attack, and the patient's condition was practically normal within a few hours. Five days later a third attack occurred, and promptly yielded to a single injection of morphine and atropine. Three more seizures followed at intervals of a few weeks, after which the patient had no further attacks of this sort. He subsequently developed a fresh cardiac infection with pericarditis, and eventually succumbed to this. Repeated examinations of the urine showed either an absence of albumin or mere traces, and sometimes a few hyaline casts, but more often none at all.

Case V.—J. G., aged sixty-one years, had first come under my observation early in 1908, after an attack of cardiac palpitation and dyspnœa which had come on during the night. He was sent to the University Hospital, where he has been a patient off and on up to the present time. Examination showed evidences of general arteriosclerosis and chronic interstitial nephritis. The peripheral vessels are sclerosed and tortuous. The blood pressure has varied from 200 to 240 mm. systolic, and 130 to 145 mm. diastolic. There is more or less constant polyuria, and the examinations have shown traces of albumin with hyaline and granular casts. Since his first admission to the hospital there have been a number of attacks similar to the one he described as having occurred before admission. The first of these which occurred in the hospital is described as follows: "The patient was suddenly seized with an attack starting with eructations, followed in a short time by coughing and expectoration of frothy material. Dyspnœa became very marked, making it impossible for him to breathe unless sitting up in bed. A hypodermic injection of morphine caused a relief, but a return of the attack required a second injection." All the subsequent attacks have been similar to this. Their onset has been sudden and without marked premonition. The lungs quickly become moist and breathing seriously oppressed. A hypodermic injection of morphine with atropine quickly terminates the seizures.

On account of the renal condition, the patient has had constant hot packs at intervals of two or three days, and his general condition has remained practically stationary. Lately, however, a slight increase in the frequency of the attacks has been noted, and their sharp demarcation from his average condition has been less distinct, as the breathing remains somewhat oppressed and the lungs slightly congested between the seizures. Some tests as to the relative value of injections of morphine and of morphine and atropine in the recent paroxysm seem to show a moderate advantage in the latter over the former.

The progress of this case has been from a condition of practical comfort for long stretches punctuated by sudden intense seizures, from which the patient quickly returned to his usual condition, to one of more frequent but less severe attacks with a more lasting state of pulmonary congestion and œdema between the paroxysms.

ETIOLOGY. The explanation of paroxysms of sudden pulmonary œdema may be sought (1) in a study of the conditions in which such paroxysms have been encountered, and (2) in the experimental and physiological data bearing upon the question.

1. *Associated Pathological Conditions.* Several authors have summarized the literature of acute pulmonary œdema, and have made tables of the various conditions with which it was associated. Samuel West[9] mentions as a rough classification of the causes of acute œdema: (1) Acute inflammation; (2) acute collateral fluxion; (3) sudden mechanical obstruction to the vessels; (4) acute fevers; (5) certain poisons; (6) hyperpyrexia; (7) acute nephritis; and (8) paracentesis thoracis; and in the detailed discussion of these groups speaks of various diseases which fall under one or the other of the headings. Riesman[10] mentions, among the associated conditions in reported cases, arteriosclerosis, Bright's disease, heart disease, asthma, acute infectious diseases, pregnancy, paracentesis of the thorax and of the abdomen, angioneurotic œdema, obscure conditions of questionable relationship (hysteria, etc.).

It is evident that the acute œdema which occurs in some of these conditions is not strictly comparable to the paroxysmal type which is under discussion. Thus, the sudden œdema occurring after paracentesis or in some violent infection, such as scarlet fever, very probably results from different causes than those which are operative in the cases in which one paroxysm after another has occurred more or less spontaneously. To say the least, there must be an added factor of etiology that determines such recurrences of œdema, and more probably the etiology is entirely different. In the case of infectious acute œdema, for example, it is very probable that the direct action of the microörganisms upon the pulmonary vessels and the adjacent tissues is the immediate cause, and it is inconceivable that repeated attacks of paroxysmal œdema are due to repeated acute infections. The same thought applies to the probable mechanical disturbances operative in the œdema following paracentesis. It is necessary, therefore, to distinguish the conditions with which acute œdema has been associated as a recurring paroxysmal event from those in which there have been only single attacks. Little help in the direction of solving the question of the etiology of the œdema in the paroxysmal type of cases can

[9] Diseases of the Organs of Respiration, 1902, i, 242.

[10] AMER. JOUR. MED. SCI., 1907, cxxxiii, 88; Trans. Assoc. Amer. Phys., xxi, 155.

be expected from mere tabulations of all the pathological conditions in which any sort of sudden pulmonary œdema occurred; but in studying the detailed descriptions of the cases which have been reported and upon which the tables are based, the paroxysmal type stands out very prominently. Taking this group alone, it is found that practically all of the cases were instances of cardiac or renal disease, or arteriosclerosis. Young persons without any of these conditions may suffer infectious pulmonary œdema, collateral œdema, or œdema following paracentesis; but the paroxysmal and recurring type has been found almost if not quite exclusively in association with the three conditions named.

So far as my own experience has been concerned, the cases have, with but one exception, been instances of cardiac, renal, or arterial disease attended with high blood pressure, and in the one exceptional case there was a cardiac lesion (mitral stenosis), and each attack of œdema was preceded by a nervous disturbance which doubtless temporarily elevated the systemic blood pressure. In some of the reported cases of recurring pulmonary œdema, unassociated with any of the three pathological conditions referred to, there have been other conditions—pregnancy (Riesman), nervous conditions (Levallée, Levy)—in which temporary or more continuous high pressure may have occurred.

The conditions which stand out most prominently in the cases that I have encountered have been increasing myocardial weakness and high arterial pressure. The occurrence of pulmonary œdema has, therefore, been more common in cases of myocardial than of valvular disease, and in cardiac disease associated with nephritis and arteriosclerosis than in the absence of such an association. The possible significance of these facts may be left for further discussion until the data of experimental investigation have been detailed.

2. *Experimental and Physiological Data.* Professor Wm. H. Welch,[11] in 1878, published the results of some experimental studies which have thrown a great deal of light on the pathogenesis of sudden pulmonary œdema. After reviewing various earlier theories he reports his experiments, which were directed to the determination of the possibility of producing pulmonary œdema by obstructions to the outflow of blood from the pulmonary circulation. Some preliminary experiments showed that the adaptability of the heart and circulation is so great that ligation of several of the large branches of the aorta failed to interfere with the work of the left ventricle sufficiently to lower the systemic circulation notably or to occasion pulmonary congestion and œdema. Ligation of the root of the aorta itself or clamping the pulmonary veins did occasion such results, but these procedures cause violent obstructions

[11] Archiv f. path. Anat. und Phys, 1878, lxxii, 375.

of the circulation such as have no analogies in ordinary disease. The crucial experiment, to which various less convincing ones led up, was one in which he was able to cause acute œdema of the lungs in rabbits by squeezing the left ventricle between his finger and thumb, thus partially paralyzing it. He concluded that acute pulmonary œdema is due to "a disproportion between the working power of the left ventricle and of the right ventricle of such a character that, the resistance remaining the same, the left heart is unable to expel in a unit of time the same quantity of blood as the right heart." He did not then deny the possibility of another explanation for collateral fluxion with œdema or for infectious œdemas, and in a recent statement (quoted by Meltzer, 1904) specifically admits that a larger proportion of the cases of pulmonary œdema than is generally supposed is due to inflammatory changes in the vascular walls. His earlier experiments, however, conclusively demonstrated the fact that a lessened output of blood from the left ventricle as compared with the right could occasion acute pulmonary œdema. It is of interest to note that in cases of marked interference with the systemic circulation, even involving the distribution of several of the large branches of the aorta, he did not obtain the same result, nor even a notable falling off in arterial pressure, a fact which can be explained only by the reserve power of the heart and the extraordinary capacity for adjustment of the vasomotor apparatus of the systemic circulation. This fact, I believe, has important clinical bearings in connection with the cases of paroxysmal œdema. It cannot be referred to any vasomotor adjustment in the pulmonary circulation, for the reason that the ligatures established mechanical obstructions in the peripheral circulation which, if not counteracted in the remaining peripheral circulatory pathways, must of necessity have caused eventual overfilling of the lungs despite any activity of the pulmonary vasomotors, if any such exist.

Sahli[12] criticised Welch's results adversely, giving two principal objections: (1) That in human cases of sudden œdema the pulse does not indicate paralysis of the left ventricle; and (2) that the pathological condition of the lungs is not that of congestive œdema. As to the first objection, Welch has pointed out that he had not claimed that complete paralysis of the left ventricle is necessary to the production of the result under consideration, and thinks that Sahli is in error in assuming that such a degree of paralysis is essential. A consideration of the physiological principles involved will easily prove the correctness of Welch's contention. Taking Tigerstedt's figures (and these are smaller than those given by other physiologists) for the average output at each systole of the right and left heart, 50 to 100 grams, and assuming that the

[12] Archiv f. exper. Path. und Pharm., 1885, 19.

left heart has lost one-twentieth of its working power, the result would be a retention in the left heart, and eventually in the pulmonary circulation of from 2.5 to 5 grams after each heart beat. In one minute, at a rate of 80 per minute, the amount of blood retained in the lungs would be from 200 to 400 grams. It is easy to see that the result must be a speedy overfilling of the lungs. That such a condition of affairs should take place in human disease presupposes that the vasomotor mechanisms of the peripheral circulation had for some reason become ineffective. A study of the cases in my series and others reported in the literature, shows that there is good ground for believing that such an inefficiency of the vasomotor mechanism had become established.

In answer to Sahli's second objection that the anatomical conditions of the lungs are not those of congestive œdema, Welch refers to his own microscopic studies in human cases which showed the existence of congestion, and notes that Sahli apparently relied on the pallid macroscopic appearance of the lungs, having seemingly made no microscopic examinations. Welch further states that microscopic examinations of lungs of pallid appearance often show marked hyperemia and diapedesis of red cells. I can confirm this statement from some recent studies of highly œdematous but pallid lungs after adrenalin injections. While denying the force of Sahli's contention so far as the acceptability of Welch's theory is concerned it may perhaps be true that the degree of œdema in some cases of acute pulmonary œdema of human beings is out of proportion to the degree of congestion. An increased permeability of the pulmonary vessels might perhaps be the explanation of a ready leakage of serum under conditions of but moderate overfilling of the pulmonary circulation. This point will be referred to later on.

Grossmann[13] and Löwit[14] also criticised Welch's conclusions, though there is no fundamental difference in the results of their experiments and Welch's. Grossmann caused pulmonary œdema by poisoning with muscaria, and found the left ventricle in a state of spasm with resulting venous hyperemia of the lungs. He believes these conditions contrary to what Welch had stated in his conclusions, having overlooked the fact that disproportion in the working capacity of the left and right ventricle and not loss of power in the left ventricle was the important fact. Spasm of the left ventricle (though according to Welch unlikely in human cases) could, therefore, produce the same results as loss of ventricular power. Löwit's experiments seemed to him to show that increased flow of blood into the lungs as well as diminished outflow are two necessary conditions. If this were correct, there would be no conflict with Welch's postulates, but other experi-

[13] Ztschr. f. klin. Med., xii.

[14] Ziegler's Beiträge, xiv, 401.

menters did not find an increased flow into the lungs a necessary condition.

A study of the experiments of Welch, as well as of those who believed their results pointed to different conclusions, seems to show without question that acute pulmonary œdema may be caused by mechanical congestion of the lungs the result of disproportionate work of the right and the left ventricles. It has been mentioned before that increased permeability of the pulmonary vessels may be a subsidiary cause, and two theories regarding the development of such a condition may be mentioned. According to some authors (Bouveret, Dieulefoy, Riesman), toxic agents acting in some obscure fashion or through the vasomotor system occasion, in cases of nephritis, rapid œdema of the lungs somewhat in the same way as œdema of the glottis occurs in the same disease. So far as the vasomotor system is concerned, it is important to observe that physiologists are practically unanimous in denying the existence of such a mechanism in the pulmonary circulation. If any exists, its range of action is extremely limited and is certainly too restricted to permit of its having more than a subsidiary place in the etiology of pulmonary congestion or œdema. So far as the second possibility, that of some direct toxic increase of vascular permeability, is concerned, analogies drawn from studies of œdema in other parts of the body make it probable that such a factor is of some importance. The fact that the lung is in a measure an excretory organ, and possibly liable to injury when vicarious excretion takes place, must be considered as having weight.

Hewlett[15] quotes his colleague Martin in confirmation of his belief that protein fluid of the concentration seen in one of his cases (total solids, 6.72 per cent., of which 6.302 per cent. was protein and 0.418 per cent. salts) could not have been forced through the vessel walls by any pressure short of one likely to cause actual rupture of the walls. However the facts may be as to this case, direct experiments have sufficiently demonstrated the possibility of œdema mechanical in nature to the extent that any cause save the mechanical element was a negligible factor. This does not obviate the possibility that some injurious agent acting upon the vascular permeability may be a subsidiary factor in cases of spontaneous œdema in man.

This is in conformity with Læber's[16] experiment, in which it was shown that the cornea with an intact epithelium of the membrane of Descemet will stand a pressure of 200 mm. of mercury, whereas once that epithelium is removed, solutions readily filter through. Very similarly, Tigerstedt and Santesson[17] found that the freshly

[15] Intercolonial Med. Jour. Australasia, 1908, viii, 611.
[16] Archiv f. Ophthal., 1873, xix, 125.
[17] Bighang. till K. Svensk. Vet. Akad. Stockholme, 1886, xi, No. 2.

removed lung of a frog filled with 0.6 per cent. NaCl solution will stand a pressure of 14 mm. of mercury for several hours without any escape of the contained fluid, whereas the same lung killed by slight heat or by pouring in distilled water, or frog's bile, at once allows filtration.

In view of these facts, it may be accepted as probable that some increase of vascular permeability may be a subsidiary cause in cases of recurring pulmonary œdema, but it would seem unlikely that such a state of affairs could develop suddenly and as suddenly subside. A more reasonable view is that a temporary pulmonary engorgement acting in conjunction with an increased or established vascular permeability is the important cause.

The facts that have brought me to the conclusion that the mechanical element is predominant are the following: (1) Welch and others have demonstrated that sudden inadequacy of the left ventricle may in a purely mechanical fashion occasion acute pulmonary œdema by causing pulmonary congestion; (2) cases of recurring œdema have generally been cases in which there have been persistent high arterial pressure and increasing myocardial weakness; (3) the attacks are sudden in onset and usually of relatively short duration, following excitement in many instances and usually occurring at night; and (4) renal disease or other ascertainable sources of toxemia may be wanting in some cases.

The first of the points has been sufficiently discussed. As to the second, I wish to add that in the majority of cases under my own observation there has been a continuous high pressure, sometimes excessively high, and prior to the onset of the paroxysms of œdema evidence of failure of the left ventricle. Among these evidences may be noted some increase in the size of the heart (dilatation) and weakening of the apex impulse and heart sounds, cardiac irritability indicated by extrasystoles, and oppression or anginoid attacks. Later, sometimes as a result of fatigue or excitement, an attack of acute œdema of the lungs occurred, and following this repeated paroxysms at irregular intervals. In the cases in which excitement or effort preceded the attacks there has been primarily a violent paroxysm of palpitation, then weaker action of the heart and œdema of the lungs. This sequence of events was clearly distinguishable in several attacks I have witnessed. These events may be interpreted as indicating a sudden extra strain imposed upon a heart struggling for a long time against excessive peripheral pressure and deprived of the protection of an intact vasomotor mechanism. Sudden external stress in normal individuals is neutralized by the inherent reserve power of the ventricle and the capacity of the peripheral vasomotor mechanism to open the external vascular channels. In cases of long-standing high pressure, arterial disease, and myocardial weakness, the cardiac power is deficient and the vessels are incapable of prompt

dilatation. Another explanation is possible in some cases, that is, that inadequacy of the coronary circulation may occasion sudden insufficiency of the heart muscle under physical strain or at times of naturally lowered cardiac power, as during sleep, without the coöperative effect of high pressure and failure of the peripheral vasomotor mechanism. Romberg[18] refers to such a condition of affairs in discussing attacks of cardiac asthma which he does not distinguish fundamentally from those of recurring pulmonary œdema. It is unnecessary to enter very fully into the question of such a distinction here, but I wish to state my entire agreement with Romberg's view. It is impossible to draw a sharp line of demarcation between cases of cardiac asthma and those of acute pulmonary œdema. Few of the former are unattended with œdema of some degree, and none of the latter are free of dyspnœa of the same type as occurs in typical cardiac asthma. The difference is one of degree only, and attacks of the two sorts may alternate in the same case.

Regarding the symptoms and etiology of cardiac asthma, Romberg states: "The cause of asthmatic attacks is generally a sudden weakness of the left ventricle, which causes intense congestion of the lungs and thus leads to extreme dyspnœa. The ventricular weakness is indicated also by marked rapidity, softness, and smallness of the pulse and frequently by irregularity and in the occasional development of dilatation of the heart. The congestion of the lungs is frequently followed by the exudation of œdematous fluid into the alveoli. This pulmonary œdema, which is indicated by small and large moist rales scattered over small or considerable areas of the lung, is a further occasion for dyspnœa. The patient often coughs up œdematous fluid which may be light or dark red or even brownish from admixture of blood and may be of considerable amounts in some cases.

"The sudden loss of power in one of the sections of the heart may be occasioned by sudden embolic occlusion of one of the large branches of the coronary artery, but is practically only met with in cases of coronary sclerosis or in cases of widespread cardiosclerosis involving the distribution of blood to numerous capillaries, even though this interference be of but a moderate grade. One may, perhaps, conceive of the attacks as due to the following condition of affairs: When the power of the heart fails or when, as a consequence of overexertion or during rest as in deep sleep, the tension in the coronary arteries falls below a certain point, the existence of sclerosis of the coronary vessels may occasion an inadequate supply of blood to the cardiac capillaries, as a result of which there must be great loss of muscular power."

While I cannot subscribe to this statement in all particulars, and especially to the view that attacks of cardiac asthma or acute pul-

[18] Krankheiten des Herzens, second edition, p. 82.

monary œdema are in the great majority of cases due to coronary sclerosis or cardiosclerosis affecting the cardiac vessels, the effect of exertion, exhaustion, and deep sleep on cardiac power seems to me undoubtedly of prime importance in the determination of attacks of acute pulmonary edema in those predisposed to such paroyxsms by long-existing high arterial tension and myocardial weakness. Romberg's statements, however, indicate another possible source of such attacks in cases in which high peripheral tension has not necessarily been a predisposing factor.

In attempting to reach some conclusion as to the relative importance of toxic increase of permeability of the pulmonary blood vessels and of mechanical engorgement of the lungs resulting from deficiency of the left ventricle, I am especially impressed by the features of such cases as Case II of my series. In this case a woman with uncomplicated mitral stenosis and without evidences of renal disease has on a number of occasions during a period of ten or more years suffered attacks of sudden pulmonary œdema, most of these attacks having followed some emotional excitement. There was never any acute illness before the seizure, and the recovery on all occasions has been so speedy and complete that within twelve hours all signs have vanished. The closest inquiry has failed to indicate even the possibility of a gastro-intestinal toxemia from dietary indiscretions. The mechanical element alone remains in evidence. One could, of course, conceive of vasomotor influences if there were sufficient grounds for believing that an active vasomotor mechanism is present in the pulmonary circulation. In other cases in which a renal lesion existed, and in which on this account toxic influences require greater consideration, the rapid onset and subsidence of the pulmonary œdema are strongly opposed to the assumption that increased permeability of the vessels as a result of toxemia is a prime factor in determining the rapidly appearing and disappearing pulmonary lesion. On the other hand, a temporary weakening, under mechanical stress, excitement, or other influences, of the left ventricle, followed by restoration of equality of action of the two ventricles, could readily account for the event. The conclusion which I have reached is that the conditions outlined by Welch in his original paper are the important ones in cases of recurrent or paroxysmal œdema of the lungs.

Treatment. The prompt effect of a hypodermic injection of morphine in cases of cardiac asthma led me to use the same remedy in one of the earliest cases of paroxysmal pulmonary œdema that fell under my notice. The result was so striking that I have since pursued the same plan, and have found this so satisfactory that it has never thus far been necessary to employ any other remedy except as an adjuvant. Some of the cases in the present series had large numbers of attacks, and eventually died, but there never was any difficulty in controlling the attacks themselves with mor-

phine alone or supplemented with other less important drugs. When the fatal termination occurred, it usually resulted from gradually increasing failure of cardiac compensation, as in Case I. In some of my cases atropine was combined with the morphine in the hypodermic injections, but on repeated occasions morphine alone was used to test the matter and was found quite as efficient as with the addition. Therapeutists have uniformly recommended atropine to cause a drying of œdematous conditions of the lung, and the drug has, therefore, suggested itself, although it is difficult to appreciate in what manner such an effect could be brought about. When it was used independently of morphine, I found that its effects were not striking, as, for example, in one of the cases in this series in which large doses of atropine were administered during the first attack and the duration of the seizure was eight hours. In a number of subsequent attacks of apparently equal severity, a single injection, or at most two injections of morphine, with small amounts of atropine, sufficed to break up the seizures within an hour. In a few instances it seemed to me that the injections of morphine with the addition of small amounts of atropine acted more satisfactorily than injections of morphine alone, but I have come to the very positive conclusion that the atropine is of very secondary importance, notwithstanding the fact that in the discussion of Riesman's paper by the Association of American Physicians[19] a number of those who took part spoke most favorably of this remedy, none, however, making any detailed reference to cases. The impression I gathered was that the remarks made were more or less off-hand and based upon the traditional applicability of this drug.

Among the few authors who have mentioned morphine as a remedy for cases of paroxysmal œdema, the most enthusiastic is Hewlett, whose experiences coincide exactly with my own. In one of his cases he used morphine followed by atropine, but found that the latter had no noticeable effect. In eight remaining attacks in the same patient he used morphine alone, and found the patient invariably comfortable and out of danger in from one-half to three-quarters of an hour. In another of his cases, during his absence, a medical colleague was called in and employed various restorative measures with little effect. Upon Hewlett's arrival, three hours after the onset, he immediately injected one-quarter grain of morphine, and within a half hour the patient was comfortable, quiet, and of good color.

The manner of action of morphine in these cases cannot perhaps be fully explained. There is undoubtedly in many of the patients an element of shock and mental disturbance which contributes to the severity of the symptoms and which may be mitigated by

[19] Trans. Assoc. Amer. Phys., 1906, xxi, 170.

the injection of morphine. This view was expressed by Billings in the discussion of Riesman's paper. My experiences, however, in the treatment of many attacks in a number of patients leads me to doubt whether this explanation suffices for the very striking results obtained. If one may draw conclusions from cases of cardiac asthma in which the results of injections of morphine, with or without atropine, are so well known, and in which an element of bronchial spasm seems to exist, one may perhaps suspect that, though it is not so apparent, a similar condition of muscular spasm is also present in the cases of pure œdema, and that by relief of this, improvement is initiated. This might readily enough explain the cessation of active dyspnœa, but could not so well explain the rapid disappearance of the œdema itself. Having regard to the pathogenesis of paroxysmal pulmonary œdema and especially its probable dependence upon relative weakness of the left ventricle, the explanation of the value of morphine may be that it exercises a direct effect upon the heart itself, quieting and steadying it; and also may have a beneficial influence by quieting restlessness and relieving muscular spasm of the extremities which increase the flow of venous blood to the right heart and thus occasion a larger supply to the pulmonary circulation. It must be confessed, however, that no explanation that can be offered at present seems entirely adequate, and the value of the remedy must rest very largely upon clinical experience. A few experiments which I have made seem to confirm this. In these experiments I injected adrenalin solution into the ear vein of rabbits, causing pulmonary œdema within a few minutes. In some of the animals, injections of morphine were made immediately after the adrenalin and in others a similar injection of atropine was made following the adrenalin. In the cases in which morphine followed the adrenalin the onset of the œdema was delayed and its intensity decidedly less, though the animals eventually developed œdema and died. In the cases in which atropine was used there was an immediate onset of œdema, which was much more decided than in the cases in which adrenalin alone was used.

Chloroform was used by Lissaman in his remarkable case in which seventy-two distinct attacks occurred. He found that he could control each of these in a very short time by inhalations of chloroform, finding that within ten to thirty minutes the patient became comfortable and was soon completely relieved. The effect of his treatment adds some confirmation to the opinion that the usefulness of morphine lies in its quieting effects. So far as the use of chloroform as a routine measure in such cases is concerned, it seems hardly likely that practitioners will generally be tempted to employ this remedy. The possible dangers of this drug in cardiac disease are too apparent to require further discussion.

Nitroglycerin has been frequently employed, and upon theo-

retical considerations may be regarded as indicated. If the pulmonary œdema is the result of a partial or considerable failure of the left ventricle, vasodilatation under the influence of nitroglycerin would undoubtedly prove useful by rendering the residual ventricular power more efficacious. In cases of cardiac asthma with moderate pulmonary œdema this result is not infrequently observed, but in the more serious cases of paroxysmal œdema my experience has been that nitroglycerin and the nitrites generally fail entirely. Possibly the explanation hinted at before may explain this failure, viz., that paroxysmal œdema occurs in cases of persistent high pressure and arteriosclerosis in which the vasomotor mechanism has been exhausted to the extent that vasodilatation, under the influence of drugs or as a result of natural reflex agencies when stress is brought to bear upon the left ventricle, has ceased to be available.

Venesection has been highly lauded, and naturally suggests itself when one observes a case of severe œdema with peripheral cyanosis. Merklen and Riesman advocate this plan of treatment above all others. Abstraction of blood from the venous system may undoubtedly relieve the pulmonary engorgement by diminishing the amount of blood flowing toward the right heart, and very probably it is in this manner that its usefulness is explainable. Compared to the simplicity of a hypodermic injection of morphine, venesection is a procedure of considerable gravity, and its effect cannot be more prompt or satisfactory.

After the first stage of the attack has been controlled by an injection of morphine and atropine, it is desirable to make use of cardiac stimulants to revive the power of the left ventricle and to promote vasodilatation, if this be possible. For these purposes injections of strychnine, digitalin, and nitroglycerin may be employed, and aromatic spirit of ammonia or brandy may be given by the mouth if the patient is able to swallow.

The after-treatment in cases in which one or more attacks have occurred consists in strict regulation of the daily life of the patient to avoid fatigue, excitement, cold, and physical strains, and some care of the diet so as to prevent overloading of the stomach and superalimentation, particularly at the evening meal. Renal cases may require warm or hot baths and particular care of the diet, and when myocardial weakness is evident after attacks, moderate cardiac stimulation may with advantage be used more or less continuously. On account of its action on the coronary circulation and on the kidneys, caffeine is a drug that may be particularly useful at this time; but strophanthus, digitalis, or nitroglycerin may be desirable.

In some of the cases under my care who had the constant attendance of a nurse I have left directions for (1) the immediate administration of aromatic spirit of ammonia upon the first suggestion of an attack, to be followed by (2) an injection of morphine

sulphate, $\frac{1}{4}$ grain, with atropine sulphate, $\frac{1}{250}$ grain, when the first definite symptoms appeared; and (3) the subsequent repetition of morphine and atropine after fifteen minutes and later injections of strychnine and nitroglycerin. This plan served to control many threatening paroxysms before they had time to become severe.

THE CONTROL AND TREATMENT OF HYPERTENSION AND ARTERIOSCLEROSIS.

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There are a number of underlying facts which the diagnostician and the therapeutist must continue to bear in mind if he would successfully prevent the results of hypertension and influence the onward march of existing arteriosclerosis. To these facts I wish to allude.

Cardiovascular, more particularly arterial changes furnish the fundamental morbid processes in the largest number of deaths after the fortieth year. We are surprised when we examine our clinical material, more particularly that seen in private and consultation practice, to note the recent enormous increase of cardiovascular disease. We are promptly forced to the conclusion when this is compared with hospital statistics that there is an increase of the diseases of the arterial tree, coronary arteries, the myocardium, and in the nerve supply of the heart, the farther we are separated from hospital practice.

A careful study of the last 3200 cases of internal disease seen in private and consultation practice offers food for reflection. These include:

Arteriosclerosis	276
Endocarditis	135
Angina pectoris	44
Hypertension	67
Mitral obstruction	62
Aortic obstruction, vascular, endocardial (inflammatory)	151
Mitral insufficiency	267
Aortic insufficiency	20
Myocardial degeneration	277
Heart hypertrophy	63
Heart dilatation	59
Senile hearts	25

In a long-hand written report of the lectures of Benjamin Rush, which has recently fallen into my possession, that erudite clinician of enormous experience and supposed unlimited clinical material, makes the statement that in all of his experience he had seen but

one case of angina pectoris, and he mentions the name of another physician practicing in Philadelphia who had also seen but one case.

Taking our clinical material into consideration, we are forced to the conclusion that hurry, worry, and excesses are at the bottom of a large majority of our cases in which we have degenerative changes in the cardiovascular system following a long or short period of hypertension. That heredity plays an important role in many of our cases we cannot deny, but we are equally positive that if these patients who are burdened with this unfortunate tendency were cautiously watched and correctly advised by the physician, changes of a degenerative nature in the heart and bloodvessels might be longer postponed. This is particularly true of arteriosclerosis as it affects the coronary and cerebral bloodvessels. It is not uncommon for us to get the history of three or four cases of coronary disease in one family, and we have family histories in which as many members of a family have died, either of cerebral apoplexy or of other brain disease associated with vascular anomalies.

These positive facts teach a valuable lesson. The study of the family history is too much neglected. Men who do not keep records, who do not question patients systematically, are very likely to overlook entirely the valuable facts which are gained by a thorough study of the patient's antecedents. These may seem rather commonplace remarks, but the consultant is so frequently brought face to face with these facts and understands so thoroughly their great importance that the opportunity to present them ought not to be neglected.

Modern pathology makes clear beyond peradventure the further fact that there is a physiological hyperplasia invading the intima during early life. We are forced to consider the separate artery as an organ, therefore, a part of the body which has positive functions to perform; that its task is not single but multiple, and that the performance of its daily undisturbed work is attended even during the earliest years of life with a compensatory deposit for the preservation of these organs and the continuity of the circulation. As soon as this physiological hyperplastic change of the intima because of age, overwork, strain, toxemia, or from any other causes is forced into a stage of hypertrophy, we have the beginning of arteriosclerosis.

There is a considerable period of hypertension which precedes this profound change in the arterial tree which, however, in the majority of cases is not recognized, because of a natural tolerance; and when present there is almost immediate compensation. If hypertension persists unrelieved and the factors which continue it remain uncontrolled, organic changes in vital organs are usually present, particularly in the heart, kidneys, and within the splanchnic area, before the patient presents subjective manifestations. In other cases, however, rather limited changes during the early days

of arteriosclerosis are sufficient to cause alarming symptoms and not infrequently prompt death. Thus, there may be early in the history of atheroma or arteriosclerosis only a small lime plaque close to the mouth of a coronary artery but of sufficient size to choke the heart without a second's warning by covering its mouth.

Hypertension long-continued is the leading cause of arterial degeneration. It must be remembered, however, that arteriosclerosis once established, hypertension is not a necessary feature; arteriosclerosis may be present for years with normal, subnormal, or high blood pressure. Hypertension upon which we fail to make a favorable impression by rational treatment is already associated with changes in the kidneys, heart, or the splanchnic area. We have failed to recognize the enormous extent of the splanchnics and their importance as pathogenical factors in the causation of hypertension and distant cardiovascular disease. The Germans have recently said, "happy is he whose splanchnics are normal and are offering no resistance to the circulation."

To prevent the baneful organic changes following hypertension we must recognize its presence early. Modern methods make this easy. Why not take advantage of these methods as we do of those which unearth kidney changes? To know exactly what resistance the heart has to overcome in the periphery is more important than to recognize the presence of albuminuria.

In considering the subject of hypertension we must remember the great clinical significance of the tightening and relaxation of the arteries of the body, the urgent need of separating the "two factors of the vessel wall and the contained blood."

Why not educate the masses in this direction that the majority may escape the ravages of final degenerative processes? When the ravages of time force the individual to seek the oculist because of changes in the crystalline lens, and this organ is flattening, and glasses are needed to compensate, or they need to be changed for this retrograde process, he has become a fit subject for blood pressure study. He is most likely in a period in which prophylaxis is demanded and it will often lead to satisfactory results. When changes are found in distant organs it must be remembered that hypertension or slightly increased blood pressure may prove an equalizer, compensatory, and ought not to be disturbed. The sphygmomanometer has proved itself a very valuable adjunct in diagnosis. It is easily and frequently misinterpreted and abused. No man should reach conclusions from the study of systolic pressure alone; if he would base treatment upon a firm basis he must think clearly, brace himself against one-sided reasoning, and give proper values to all data which are unearthed by the thorough consideration of subjective symptoms and the knowledge gained by physical examination: the sphygmomanometer furnishes but one strong link.

When we find with evidences of hypertension or already present arteriosclerosis accentuation of the aortic second sound we know that "the door is being slammed"—the cusps forming the door—that there is a peripheral obstruction which heart and artery are trying to overcome and we must act accordingly.

The point which I seek to impress with greatest earnestness is that if we would prevent arteriosclerosis we must consider the artery an organ with a capacity which is great for work, but which when abused or overworked, insulted or poisoned, revolts and begins to degenerate. When one robs the heart of its sleep he is damaging that organ and the entire vascular system. Lauder Brunton in his happy way has recently said, "the heart practically sleeps more than the brain or the body, but the great distinction between the sleep of the heart and that of the brain, is that the sleep is so short at a time." The heart and arteries demand thirteen hours of this sleep daily (this statement surprises those who have not computed the time). When robbed of this repose the periods of rest are shortened; cardiovascular organs are forced to work over-time, more rapidly than before, and the foundation for retrograde process is laid. Nature will bear insult a reasonable time; the revolt will come sooner or later with unfailing certainty.

The final break in a large number of cases includes myocardial degeneration with all of its pangs. Hypertension and arteriosclerosis are increased less by the routine work of the day than by the innumerable outside affairs and engagements which occupy so much of the time of the busy man (particularly the physician), and cut his rest to a minimum. These workers hold the string taut, the tension under which they work is enormous; their bearings are worn, until at last the string snaps and we are called to repair and reconstruct—often when it is too late.

Hypertension will often lead to a strong suspicion of renal invasion. This is true when associated with hypertrophy of the left ventricle though albumin be absent from the urine during a long period. The renal arteries may be far advanced in arteriosclerosis without invasion of other kidney structures which when changed add casts and albumin to the urine. From the careful investigation of the diseases of the ductless glands we are convinced that these have a marked influence in producing arterial change and that in considering the rational treatment of hypertension we are not to divorce ourselves from this possibility. The production of arteriosclerosis in animals by the injection of adrenalin is proof positive of the correctness of this statement and must have a decided bearing on the dietetics of hypertension and arteriosclerosis. The younger the subject the more unfavorably does he react to hypertension and arteriosclerosis. Indeed, we are often justified in speaking of the presence of arteriosclerosis in the aged as a protective process —a true paradox. If we would successfully control blood pressure

we must have the hearty coöperation of the patient. He must be willing to surrender himself absolutely to his physician. Carlye's definition of genius included infinite attention to detail. Every possible bearing of the life and habits of the individual upon these destructive and degenerative processes must be taken into account by the therapeutist, and this in detail.

Romberg says, "Everyone acquires his arteriosclerosis within the circuit which he has taxed most." Upon this truth we are to act, particularly in attending early to local disturbances, to all intestinal anomalies, including chronic constipation, intestinal indigestion, and the dyspepsias. Even a distant irritation as a urethral stricture or unrelieved prostatic hypertrophy may keep up hypertension and lead to arteriosclerosis.

Tobacco, that is, nicotine influences blood pressure very powerfully. Lauder Brunton says, "The rise of blood pressure is so great that I have never seen it equaled after the injection of any drug, with the exception of suprarenal extract. This rise is due to contraction of the arteries. The ultimate effect is to increase the rapidity of the heart. When this pulse is quickened no stimulation of the vagus will slow the heart, as its terminal branches in the heart are paralyzed by the drug." I continue to quote Brunton: "Usually tobacco is employed by smoking, either in the form of cigars or cigarettes, or in a pipe. When used in any one of these forms it is not pure nicotine which reaches the mouth, but really the products of the dry distillation of tobacco, containing a large quantity of pyridine and picoline bases. Probably nicotine in greater or less quantity is also present. The proportions of the pyridine and the picoline bases in the tobacco smoke vary according to the mode in which it is burnt. In a cigar there is freer access of air, so that much collodine and little pyridine are formed, while in a pipe much more pyridine is produced, and thus stronger tobacco can be smoked in a cigar than in a pipe. So much is this the case, that tobacco which in the form of a cigar would produce no disagreeable effect, may cause giddiness and vomiting if smoked in a pipe. The smoke from a pipe or cigar usually passes simply into the mouth and out again, either through the mouth or the nostrils; but when smoked in a huka or narghileh, the smoke is inhaled into the lungs, and this is frequently done also by people who smoke cigarettes. When a huka or a narghileh is used, the smoke passes through water before being inhaled, and it is thus deprived of most of its poisonous constituents; but this is not so with the smoke of cigarettes, and, as absorption occurs very rapidly from the pulmonary mucous membrane, cigarette smoking is sometimes very injurious. There is another reason, however, why cigarette smoking is frequently more harmful than smoking a pipe or cigar, and it is that cigarettes are small and can be smoked in a few minutes, so that many more cigarettes than

pipes or cigars are consumed in the course of a day, and the total quantity of tobacco used is thus much greater in the form of cigarettes. Smoking in moderation does not seem to be injurious to grown-up people, but there appears to be a general concensus of opinion that it is very distinctly harmful to growing lads." Whatever conclusions we reach concerning the advantages or pleasures derived from tobacco by the healthy individual, it should be absolutely interdicted in the presence of hypertension and arteriosclerosis.

Coffee raises blood pressure because of the caffeine which it holds, and this rise is associated with increased rapidity of the heart's action. It makes the heart irritable, it increases the power of the heart's contractions, it places an extra load upon the kidneys, increasing the urine flow, and the solids of the urine are increased by overtaxing its secreting cells. Because of these effects I have for some time held that men beyond fifty who get insufficient exercise or are brain workers, and children before puberty, cannot with safety to themselves drink coffee. There is something in the coffee bean besides the caffeine, of which it can be freed, which also acts as a healthful stimulant to brain and other organs and makes it a valuable article of diet. A patented process is now being used in Germany by which the coffee bean is freed of 90 per cent. of its caffeine. This coffee has been largely used abroad during the past two years with great satisfaction. There is scarcely a health resort in Germany which is not largely substituting this coffee for the ordinary coffee of commerce. The taste of the coffee is not materially changed from that of ordinary coffee, the effect, however, upon the heart and bloodvessels is decidedly different. Patients who have never been able to take coffee, whose circulation, particularly the heart, has been unfavorably affected are able to take it when freed of its caffeine with impunity. During the past year I have given it a fair trial and am thoroughly convinced of its value and the desirability of using it in all cases of hypertension, arteriosclerosis, and irritable heart; indeed, I believe that it is the ideal coffee for all brain workers or those of sedentary habits after the age of fifty. Blood pressure is not increased by its use, nor is there palpitation, or annoying systolic force.

The dietetic treatment of hypertension and arteriosclerosis is most important. No abnormalities from which we suffer are more favorably influenced by rational diet than are these conditions. The surcharged artery is always a menace to the individual. When the pabulum which it carries contains an excess of purin bodies, or of irritating substances of any kind, hypertension is increased and arteriosclerosis invited.

We have often been surprised by the routine and empiric methods which are practised in many sanatoriums, here and abroad. In some of these, patients with hypertension are treated by what is known as the "flushing method." It is supposed that by giving quarts

of water daily the noxious substances which are floating in the blood are promptly eliminated. No thought is given to the distention of vessels or to the extra work given the heart and kidneys which follows this irrational treatment. Healthy elimination must, of course, be invited but not at the loss of heart strength, nor by the overdistention of the artery or the overworking of the kidney. No physician who prefers the use of natural methods decries the influence of pure water, but we must raise our voices against its excessive and empiric use.

In connection with the subject of diet we are again reminded of the overpowering importance of the study of the individual case. The tolerance of these patients must be tested before full directions can be given. In some we find a low carbohydrate tolerance, in others, purin bodies at once choke the furnace, in still others the digestive organs are so changed as to influence unfavorably the vascular system because of motor insufficiencies, either in the stomach or the intestines. In other cases we find that an ordinary diet is keeping up a hyperacid state which promptly leads to a train of symptoms including joint changes, final gout, with associated cardiovascular disturbances. Modern methods make it possible for us to clear the horizon without great loss of time, but with great benefit to the patient.

Sufficiently reliable results for practice are obtained from the estimation of the purin bodies by the Walker-Hall apparatus, the uric acid output after the method of Rehmann, while the tests for indican, ammonia and other noxious substances are easily made by those who care to study these conditions thoroughly.

Patients with hypertension or arteriosclerosis demand continuous watching; articles of food may be tolerated at one time which for unknown reasons exaggerate symptoms and are harmful at other times. If we had our choice of treating angina pectoris or coronary disease by a single method I think we would all promptly choose the dietetic. It is surprising to note how much can be accomplished even with advanced sclerosis of the coronaries by a rational diet and proper living without the administration of a single grain of medicine. I know of no disease in which the results are so brilliant following appropriate diet as in angina pectoris. Patients who have had repeated severe seizures, who suffer from stenocardia on slight exertion, promptly yield to the diet which gives them just food enough and not too much, which withholds from them the heavier and more indigestible articles of diet, who take but little meat, never more than once daily, often for days no meat at all; but who are preferably given eggs, fresh fish, easily digestible vegetables, and a limited supply of liquids. These patients demand the minimum of food that will nourish them. Many patients with angina pectoris have promptly lost their lives because of a single dietetic indiscretion. I cannot express too

forcibly my ideas on this subject in connection with this disease which has robbed us of so many brilliant colleagues. The question of diet is of the greatest importance in connection with those cases in which there is a strong hereditary tendency.

Agreeable occupation which favors a quiet mental state is of enormous value in the treatment of both hypertension and arteriosclerosis. An exalted and overwrought brain must of necessity be associated with the tear which follows the abuse of that organ. This is, at once, associated with hypertension; prolong it and the associated pathological conditions will not be long postponed. I have recently had an example in a lawyer who was engaged in the promotion of an enterprise of enormous proportions, who was without hereditary taint, in whom the hurry, worry, and excitement, associated with the launching of this project promptly led to enormous hypertension, even thickening of the arteries, renal invasion, and death in the course of a few months. These cases are not uncommon, and while the process is not always so rapid or widespread as in the case mentioned, subjective and objective symptoms are not long postponed.

Therefore, to the busy brain worker, whether he has hypertension or not, we are forced to recommend periods of quiet, prolonged rest, change of scene, proper exercise, and temperance in all things. While alcohol may not directly cause arteriosclerosis we are very sure that it does so indirectly and that productive changes in the kidneys and other organs consequent upon its use calls upon the cardiovascular system for extra work which finally leads to change of a degenerative character. Individuals react differently to the social glass of wine or liquor. My experience has been that alcohol in any form is injurious in all cases of hypertension and arteriosclerosis except in a final stage associated with myocardial weakness and broken compensation, to which I shall again refer.

We are all praying for the prolongation of life which is to follow the introducing of the lactic acid bacillus as suggested by Metchnikoff. To those of us who have turned the corner at fifty there is cold comfort in the statement of Metchnikoff that if our lives are to be prolonged we should have taken advantage of the inhibitory action of the lactic acid bacillus during our early days. However, buttermilk is on trial as never before. Whether by suggestion or by its inhibitory action on toxin producers in the intestines, it has seemed to make light the hearts of many.

The baneful effect of excessive coitus has not been thoroughly recognized by the profession. At any rate, the hypertensive and arteriosclerotic have not been sufficiently warned against excessive indulgence. In some of our cases abstinence becomes imperative. This is particularly true of those types of vascular disease associated with arterial spasm.

We forget that the heart muscle is not the only true muscle con-

cerned in the circulation of the blood. The skeletal muscles are as much a part of the circulatory system as are the capillaries, the veins, or the lymphatics. A healthy circulation demands the assistance which it receives from the heart muscle, the arteries, capillaries, veins, lymphatics, and not the least important, the skeletal muscles. The circulatory poise is best preserved by attention to all of these separate organs. The healthy stimulation of muscle by massage, proper exercise, with sufficient periods of rest, becomes exceedingly important in the treatment of the conditions which I am considering. Cases in which active exercise is out of the question demand passive movements. The Zander movements are particularly useful. It is unfortunate that we have in this country no such institutions as we find in Germany, rivalling each other, in which it is possible by means of the Zander apparatus to stimulate most muscles of the body and to do this without danger to the heart or bloodvessels.

The introduction into therapeutics of the various devices perfected by Zander marked an epoch in mechanotherapy which has not been sufficiently appreciated by the profession. In practice many well-selected cases of hypertension and arteriosclerosis are favorably influenced by warm and hot baths. I have observed the effect of these at Nauheim, Weisbaden, and Karlsbad, and make the statement knowingly that in well-selected cases, though the practice is empiric, the results are often reassuring.

No one health resort has a monoply of hot carbonated or saline water. On the other hand, we cannot ignore the fact that the environment of success is not to be disregarded. The rest, change of scene, and divorce from active life are unquestionably factors which make it possible to give the heart, arteries, and other organs that rest which is so much needed in these cases. If the patient will yield, is willing to rest at home, the Nauheim baths with resisted movements are often used to better advantage under his own roof than in the crowded hotels or boarding houses of the health resort, or in the depressing atmosphere of seriously sick patients. The discouraging factors with busy men are that they fail to take advantage of our suggestions; do not rest absolutely, and continue the supervision of their interests from their homes, which often complicates matters and brings home treatment into disrepute.

Until within the past year I failed to recognize the value of high frequency currents for the treatment of either hypertension or arteriosclerosis. During the past summer I had abundant occasion to note the effects of this treatment in Karlsbad under the direction of Dr. Buxbaum. Formerly I attributed the relief of subjective symptoms to the overpowering influence of suggestion. I was promptly convinced, however, that this conclusion was unjust and found that this method of treatment had influenced a large number of cases favorably, that blood pressure was reduced, and

that subjective symptoms were relieved; in some of the cases the relief continued during a number of months after a sufficient period of treatment.

We are not to conclude that this is a curative agent. It does, however, materially influence blood pressure, and it does dissipate in many of these cases, the annoying subjective symptoms. In advanced arteriosclerosis, the blood pressure is not likely to be reduced. In some the pressure may be raised by the treatment. The cases which react favorably are young subjects with hypertension or those who have symptoms of incipient sclerosis; in these there is a lowering of blood pressure with corresponding improvement in the general condition. Laquer holds that in these cases the benefit is gradual but, as a rule, permanent. He also holds that when there is cardiac disease without arteriosclerosis, good results are obtained by the local application of one electrode over the precordial region. How this agent acts I do not know; that it is beneficial I am positive. I have seen cases of angina pectoris treated by this method in which subjective symptoms were held under control during long periods. I do not refer to those cases with the larger attacks, but to those in which the sternal pang and oppression were present at some time during each day.

I am sometimes asked what drug have I to recommend for the relief of the conditions under consideration. Drugs alone are inadequate to meet the indications mentioned, but there are drugs which may be given in connection with the methods of treatment already suggested which lead to material benefit and which in incipient cases may lead to cure. The preparations of iodine have for years been the sheet anchor of the therapeutist for the treatment of arterial disease. The great trouble in the average case is that the physician in giving his original prescription for the iodide fails to make clear the urgent need of long-continued treatment with these remedies. If the iodides are to relieve or cure they must be administered during months and even years. Patients who do not tolerate the potassium iodide often benefit by alternating the use of the strontium, rubidium, and sodium salts. I have found that the strontium and rubidium salts are better borne than the sodium or potassium iodides. The more valuable iodides for the control of symptoms and the influencing of the underlying process are the potassium and sodium salts. It is very easy in the average case to establish tolerance by giving the strontium or rubidium salts first; finally the sodium salts, then a long-continued period during which the potassium salt is taken, and as time wears on a return either to the sodium or rubidium iodide for a short time. The potassium salt, of course, is to be given during the longest possible period. Recently there have been introduced other preparations which have also seemed to be efficacious, and readily borne. I refer to sajodin, tiodin, and

eustenin. The profession has had a fairly satisfactory experience with sajodin. Tiodin is a preparation of iodine and thiosinamin. In occasional cases the tiodin has served very well. Eustenin has lately been introduced and is a combination of sodium theobromate and sodium iodide. It is a whitish hygroscopic powder, soluble in water. Theobromin has been supposed to increase the coronary circulation and advances the blood flow through these vessels and through the cardiac muscle; the iodides dilate the vessels and cause a dimunition of the viscosity of the blood. The combination of the iodine and theobromin in the form of eustenin has in some cases in Van Noorden's and Jargie's practice been satisfactory in relieving subjective symptoms and reducing blood pressure. A number of years ago, Huchard recommended the use of pure theobromin for the relief of sensory symptoms associated with coronary sclerosis. This drug has been given a fair trial; the majority of those who have used it are willing to confirm Huchard's observations. Theobromin is particularly useful in cases of angina pectoris, and intermittent claudication or painful vascular spasms. I have been in the habit of prescribing not more than 0.24 gram of pure theobromin every morning and night in the average case. It is a purin body and ought not to be used in too large doses. In practice I have found that larger doses are likely to cause annoying headaches. Cases in which with myocardial weakness and coronary sclerosis there are almost continuous sensory symptoms are likely to show improvement from the frequent administration of small doses of the drug; 0.12 gram may be given every two or three hours according to the urgency of the symptoms. The salicylate of theobromin in my experience is not as efficacious as is pure theobromin. Painful processes due to sclerosis, associated with myocardial degeneration, broken compensation, dropsies, in spite of the thickened arteries frequently show wonderful improvement after the administration of powdered digitalis in 0.1 gram doses with 0.6 gram of the salicylate of theobromin. I have occasionally substituted for the latter 0.24 of caffeine sodium salicylate. In these cases it is wise to combine with the digitalis treatment the daily morning administration of one tablespoonful of a saturated solution of Rochelle salts or magnesium sulphate in one-half gobletful of hot water before breakfast. This will empty the splanchnics and act as a safety valve. In these cases because of extreme weakness an occasional glass of claret, Tokay, or even a small dose of whisky, or ethereal stimulant, preferably the etheral tincture of valerian, will brace the patient and carry him over a critical period until the other remedies already suggested come to his rescue.

Persistent hypertension treated with the diet and methods of living suggested is often favorably influenced by the administration

of the Lauder Brunton draught; the modified prescription for which I offer:

℞—Sodii nitritis 3.0
Sodii bicarbonatis, C. P. 32.0
Kalii nitratis 32.0
Aquæ . . . . . q. s. ad. 132.0—M.
Sig.—Shake well. One teaspoonful in a gobletful of hot water before breakfast.

In rebellious cases of hypertension, usually associated with sclerosis, I give this remedy in the same dosage and in the same way before each meal. Small doses of chloral (0.3 gram) will in many cases of uncomplicated hypertension, with attention to the digestive system and thorough emptying of the intestinal tract at short intervals by means of salines and the use of alkalies, cause a decided drop of blood pressure and great relief of associated subjective symptoms.

Pounding, irritable hearts, with hypertension in which the systolic contraction is unusually strong, the area of cardiac impulse outside the nipple line, with or without vertigo, more or less discomfort referable to the head, are, as a rule, relieved by the administration during two or three weeks, three times daily, of from 1 to 1.5 gram of strontium bromide with from two to four drops of either tincture of veratrum viride or an equal dose of tincture of aconite root. The nitrites, including nitroglycerin, sodium nitrite, amyl nitrite, are most useful in those cases of arteriosclerosis associated with sensory symptoms. For the continuous treatment of hypertension I prefer the sodium nitrite with theobromin to any of the other vasodilators. For the relief of vascular spasm, attacks of angina, in which the physiological action of the nitrite needs to be prompt, nitroglycerin, erythrotetranitrate, amyl nitrite are preferable. Small doses of glonoin in extreme cases produce very little effect, only evanescent results. When we are face to face with danger in these cases only large doses of glonoin, preferably in liquid form, and not in tablets, dropped upon the tongue will produce the physiological effect which is absolutely necessary to prolong life. The lives of patients with threatening death from cardiac spasm or true stenocardia have, in my practice on several occasions, been prolonged during many years by the administration of nitroglycerin in large doses, sometimes as much as fifteen drops in a single dose.

We are not to be held by the rule which interdicts the administration of digitalis in all cases of hypertension, for occasionally, in spite of high blood pressure, there are convincing evidences of myocardial weakness without marked fibroid degeneration in which digitalis does yoeman's work. Such a case I have at present under observation; we hesitated to administer digitalis during a long period because of the serious sensory symptoms due to coronary disease, but all of these yielded promptly after the use of digitalis when the systolic strength was established.

I am fully agreed that Mackensie is correct when he makes the statement that "some of the most violent attacks of angina pectoris have occurred in people in whom the exhaustion of the heart was temporary, and the restoration of reserve force resulted in a complete cessation of pain and in permanent recovery." Some of these I have treated with small doses of strychnine and 0.3 gram doses of lactate of lime.

In the treatment of both hypertension and arteriosclerosis we receive valuable indications from the consideration of the general condition of our patients and the constitutional treatment accordingly administered. With associated renal complications and anemia the chlorides are particularly valuable. We must continue to keep before our mental vision the urgent necessity of conveying to the heart and the entire system the pabulum which it demands, including an abundance of oxygen.

The life of the Venetian Doge Cornaro who presented a discourse on "The Art of Living Long" has never ceased to interest me. It is a classic and should claim your attention. Cornaro had, I dare say, subjected himself to overwork, and possibly to many other baneful influences, in spite of which he reached his eighty-third year. He appreciated the fact that he had made many mistakes, and that he was fast nearing his end. His later experience justified the production of a series of articles, "Wherein the Author details the Method by which he Corrected his Infirm Condition and Strengthened his Naturally Weak Constitution and Thenceforth Continued in the Enjoyment of Perfect Health." He lived to be over one hundred years old, enjoying to his last day the full possession of his faculties and many other pleasures which he was quite sure he would not have enjoyed had he disobeyed the laws of Nature. A single quotation is justified: "Divine sobriety, pleasing to God, the friend of Nature, the daughter of reason, the sister of virtue, the companion of temperate living, the loving mother of human life, the true medicine both of soul and body, how much should men praise and thank thee for thy courteous gifts, for that gives them the means of preserving life in health, that blessing which it did not please God we should have a greater in this world, life and existence so naturally prized, so willingly guarded by every living man." This paragraph contains the essence of therapy which the thinking physician must apply against hypertension, whatever the cause.

THE TONIC USE OF DIGITALIS.

By Emile Schmoll, M.D.,

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The diagnosis of the disturbances of the different functions of the heart muscle has made tremendous strides under the influence of Mackenzie's graphic method of recording the heart movements and Wenckebach's ingenious application of Gaskell and Engelman's explanations of physiological phenomena. Studies in the disorders due to disturbed conductivity have opened a new chapter in the seemingly exhausted subject of the pathology of the heart. We now interpret the alternating pulse as a sign of disturbed contractility, and the disorders of excitability and rhythmicity are rapidly becoming identified. The rational therapy of the future will have to aim at the correction of these primordial disturbances, and modern pharmacology will have to rise to the demand for the various specifics. Unfortunately, tonicity, the most important function of the heart muscle from a clinical standpoint, cannot be directly recorded or estimated. Its variations must be gauged by a study of the functional disturbances due to impaired circulation. Stretching of the muscle and distention of the ventricular cavities follow necessarily on a loss in tonicity, and the result is a retardation of the blood stream.

The influence of the tonus on the efficiency of the heart muscle may be appreciated from the following observations: It has been shown that the ventricle does not empty itself completely with each systole. It fills during diastole with a varying amount of blood, depending mainly upon the length of diastole and the strength of the forces which aspirate the blood into the heart. We know also that a muscle can do most work when it is slightly stretched. Thus, with a good tonicity, the strength of contraction of a given heart muscle will vary directly with the amount of diastolic filling of the ventricle. Also, as the capacity of the ventricle varies as the cube of the diameter, less shortening of the muscle is needed to expel a given quantity of blood from a well-filled than from a poorly-filled cavity. If, however, the stretching of the muscle be due to a loss of tonicity, even an increase in contractility cannot prevent a loss in the amount of work done. This can be shown in the laboratory by loading a muscle and stretching it beyond the limit of its tonicity. In this condition it can perform very little work.

There is still another way in which a loss of tonicity interferes with the action of the heart. Under normal conditions ventricular systole begins with an increase of tonus, which raises the pressure in

the cavities to that of the pulmonary arteries and the aorta before any shortening of the muscle has taken place. Thus, with the opening of the valves, muscular contraction begins under the most favorable conditions of tonicity and load.

These conditions are changed just as soon as any one of the heart valves begins to leak. The escape of blood allows the ventricle to change its shape at the very beginning of systole, there is no rise in tonicity, and the fibers begin to contract too soon. The contraction, which should be afterloaded, becomes isotonic, that is, the amount of work performed is practically equal throughout the duration of contraction, and much more energy is used up.

The lack of isometric contraction preceding the isotonic contraction can be overcome to a considerable extent by an increased diastolic filling of the ventricle. This stretches the muscle enough to raise the tonicity appreciably before the beginning of systole. A shorter contraction is then needed to expel the usual amount of blood. This compensatory process soon tends to become harmful, as the overstretching of the heart muscle lowers tonicity still further.

It is its influence on this vital function of tonicity which has given digitalis its unique position in the therapy of cardiac disorders. As far back as 1882, François Frank showed that digitalis strengthens the muscular rings which are largely responsible for the competency of the auriculoventricular valves. The relative mitral insufficiency seen in various conditions of depression—anemia, cardiac overstrain, and failing compensation—is due to a loss of tone in this ring, and can be influenced favorably by digitalis. Since Frank's publication, numerous investigators, notably Cushny and Cameron, have come to the same conclusion, and Gossage has tried to prove that this effect on tonicity constitutes the entire beneficial action of digitalis. This view, however, is probably not wholly justified. Balfour[1] says: "All the benefits we obtain from digitalis are inseparably connected with its tonic action; they flow from the power that digitalis has of increasing muscular elasticity, and the improved metabolism of all the tissues, but especially of the myocardium."

The effect of digitalis upon the heart depends upon the amount of the drug present in the body. In fully developed heart failure, with dilatation, enlargement of the liver, and œdema, we give digitalis in massive doses. Accumulation takes place owing to the comparatively slow excretion, and the body becomes saturated. Then appear the toxic as well as the beneficial effects of digitalis. The slower pulse and longer diastole, due to vagus action, permit a more complete recuperation of the contractile power, and the deep and more forceful inspirations, due to stimulating of the respiratory centre, aid in the diastolic filling of the ventricles. The dose of digitalis

[1] Clinical Lectures on Diseases of the Heart, p. 360.

must be adapted to the rate of its excretion if we want to get the effect on tonicity alone. This rate has been found by Balfour to be about 1 grain in twelve hours. Accumulation does not take place with this dose, and the drug may thus be given over periods of weeks, months, and even years without any toxic action.

This valuable point in the art of giving digitalis has received scant mention in the literature of the subject. Most of the experimental work has been done on normal animals, and with toxic doses. In this way many errors have been introduced into the clinical application of digitalis; for instance, the action on the peripheral vessels which appears only with toxic doses.

Cloetta[2] has made some excellent studies in this use of "tonic" doses in contradistinction to the "anti-asystolic doses" of Huchard. First, he took normal animals and gave digitalis in increasing doses for periods varying between a few months and two years. There was no change in the pulse frequency, and measuring the blood pressure during compression of the aorta showed no increase in the ability of the heart to overcome the obstruction. Autopsies showed no change in the weight or size of the heart. The result of the experiment was very different, however, when performed on animals with an aortic insufficiency produced by the Rosenbach method. The hearts of the control animals which received no medication showed an increase in weight of 47 per cent. over the normal average, while the hearts of the treated animals showed an increase of only 25 per cent. The work-output of the digitalis heart muscle was vastly greater, however, than that of the controls.

Four of the control animals died of heart failure, while none of the treated animals showed any symptoms of heart disease. The functional test—blood pressure during compression of the aorta—showed practically normal records with the treated, and much lower figures with the control animals.

We see from these experiments that the larger hypertrophy gives a poorer functional result. This can probably be explained in the following way: The loss of tone is a stimulus to hypertrophy, which, however, cannot make up entirely for the original loss. Digitalis helps the muscle to keep its tone; less hypertrophy is demanded, and the new muscle is again enabled to work more effectively.

With the great development of our modern methods of physical diagnosis, the tendency has been to rely too much on the direct examination of the heart. As stated at the beginning of this paper, we must look elsewhere for the first signs of failing tonicity, and if we wait for indications from the heart itself, we will need the massive doses to get out of danger. Neither will blood pressure determinations help us, as they offer no means of measuring accurately the systolic output of the heart. Plesch's[3] procedure, which seems to be

[2] Archiv f. exp. Path. und Pharm., lix. [3] Ztschr. f. exp. Path. und Ther., vi.

the only one giving any degree of accuracy, is hardly applicable clinically.

Loss of tonicity shows itself first in general symptoms. In the presence of heart disease, persistent cough with rales over the basis of the lungs may be attributed to a loss of tonicity of the left ventricle. A similar condition on the right side of the heart shows itself first in the swelling of the liver, dyspeptic symptoms, and slight pitting of the ankles, which disappears over night. When these symptoms coincide with the appearance of a mitral or tricuspid murmur, all indications are present for the tonic doses of digitalis.

The tonic use of digitalis in cardiac disease is of comparatively recent introduction. The cumulative, toxic effect from repeated small doses seemed so inevitable as to prohibit any such continued medication. Traube was the first to see a way out of the difficulty, and he showed that digitalis could be used over long periods in slowly increasing dosage. A little more attention was paid to the subject when Baelz[1] reported the remarkable case of a woman who had taken an infusion of digitalis for seven years. While taking the drug she was able to work, but any attempt to stop was followed by diminution in the amount of urine, œdema, flashes of light, tinnitus aurium, and subjective heart symptoms, which would disappear within a few hours after resuming treatment. In the course of seven years she took 810 grams of digitalis.

Balfour in England, Fraentzel[5] in Germany, and Huchard[6] in France have been pioneers in this field.

Balfour, in his lectures, discusses the subject so fully and so well, that very little has been added since. More recently, Groedel,[7] Naunyn,[8] and Kussmaul[9] have insisted on the advantages of this method. Kussmaul has given us, in his usual classic style, a most complete and accurate account of a case in which autopsy showed the following conditions: Generally dilated heart, stenosis and insufficiency of the aortic valves, aneurysm of the aorta, arteriosclerosis and endarteritis of the coronaries, and contracted kidneys. He first took digitalis in 1892 on account of œdema and dyspnœa, and from that time until his sudden death in 1899 he continued its use in daily doses. During that time he travelled extensively, and was in fairly good health. Occasionally, after some unusual strain the dose had to be increased for a few days to obtain satisfactory diuresis, but the protective action of digitalis gave him a comfortable range of effort, and he suffered from none of the usual phenomena

[4] Archiv f. Heilkunde, 1876.
[5] Charité Annalen, 1868, Vorlesungen über die Krankheiten des Herzens.
[6] Les Maladies du Cœur.
[7] Verhandlungen des Congresses f. innere Medicin, 1899.
[8] Therapie der Gegenwart, 1899.
[9] Ibid.

of heart disease. According to accurate records kept during the seven years, he took in that time 305 grams of digitalis.

As these are the only publications I can find on the use of digitalis in tonic doses, I feel justified in communicating my experience in this subject, collected from a large number of cases observed over a period of several years.

Looking over the records of these cases, I can distinguish two groups which have different prognoses and treatment. In the first group are the valvular lesions of rheumatic origin. It is principally in these cases that we derive the full benefit from the tonic action of digitalis. Then, the lesion once established, the heart has to overcome a certain constant resistance. An alteration in the compensatory mechanism is demanded only with an inter-intercurrent infection, rheumatic or otherwise.

In the second group are the myocardial and arteriosclerotic cases, in which the pathological processes are progressive. The heart has to increase its work constantly to compensate for the increasing loss of tonicity, to keep up excretion in the contracting kidney, and to overcome the high blood pressure.

Let me first consider the valvular lesions. In my series these are all at the mitral valve, and in most of them the stenosis is the preponderant factor. A typical example may be found in the following case:

Case I.—Mr. M., aged forty-two years, came under my care on September 19, 1908. He had had two attacks of rheumatic fever fourteen and eight years previously, and five years ago he had an attack of gonorrheal rheumatism. Ever since that time he has been conscious of his heart, and gets out of breath easily. Nevertheless, he continued his usual mode of life, filling a very responsible and strenuous position by day, drinking and smoking very heavily at night. In December, 1907, he had an attack of influenza, which left him weak and not fully recovered. In the following June he noticed swelling of the legs and abdomen, and dyspnœa appeared. Jaundice had developed a few days before I saw him.

A diagnosis was made of mitral stenosis and insufficiency and tricuspid insufficiency. The pulse was permanently irregular—about 140 per minute; the blood pressure, difficult of estimation on account of the irregularity, was approximately 160 systolic, and 110 diastolic (von Recklinghausen, water pressure).

The patient received 0.7 gram of digitalis in the next four days. It was then discontinued, as the pulse had dropped to 60, with the onset of an enormous diuresis. At that time an early diastolic murmur could be heard; also a distinct reduplication of the pulmonic second sound. Though kept in bed all the time, œdema reappeared on October 9, with rales over the bases of the lungs. Digitalis was resumed, 0.1 gram being given daily. The result can be seen in the following table:

Date.	Digitalis.	Fluids. (24 Hours) Ounces.	Urine. Ounces.	Specific gravity.
October 2		24	30	1018
" 3		34	34	1020
" 4		36	40	1014
" 5		36	34½	1014
" 6		36	44	1014
" 7		46	29	1016
" 8		36	35	1017
" 9	0.1	36	33	1016
" 10	0.1	42	32	1017
" 11	0.1	42	42	1015
" 12	0.1	36	49	1017
" 13	0.1	39	53	1012
" 14	0.1	36	62	1011
" 15	0.1	46	45	1012
" 16	0.1	39	36	1018

Note that the digitalis action appeared on the 11th, the third day of the treatment. The heart now seemed to be well compensated; the œdema had disappeared, and he was able to go about his business.

On October 29 some œdema reappeared, with enlargement of the liver. The dose of digitalis was immediately increased to 0.3 gram, with an increase in the diuresis and a dropping of the pulse rate to 68 next day; 0.2 gram was given that day, and the day after the usual daily dose was resumed. When seen on November 20, the heart was well compensated; there was a strong apex beat; pulse 68, no œdema. Blood pressure: systolic, 160; diastolic, .85. The pulse remained permanently irregular. On December 3 slight œdema of the legs appeared, and on the 6th the dose was again increased to 0.3 gram; 0.2 gram was given on the 7th, and on the 8th the regular dose was resumed. Diuresis and disappearance of the œdema followed on the 7th.

Since then the patient has remained well, attending to his business, and he has been fully compensated, except on two occasions, when some physical overstrain brought on swelling of the liver and slight œdema. On both occasions the disturbance yielded on the second day to the usual increase of digitalis. He has now been taking 0.1 gram of digitalis regularly every day for twenty months, and we see the typical result of such treatment. A heart which failed under the slightest provocation (October, 1908) has been toned up so that the patient has been able to attend to all of his duties without any symptoms of failing compensation.

Such experience has taught me to follow up the cumulative doses by tonic doses of digitalis in every case in which a rheumatic valvular lesion has led to heart failure. As soon as the full digitalis effect is obtained, usually on the second or third day, I give 0.1 gram and continue this as long as the patient is under my care. In this way I have kept a number of advanced cases fully compensated for many years.

As stated earlier in this article, tonic doses may be used to great advantage when slight œdema, and enlarged liver, and a decrease in the urinary excretion point to an imminent failure of compensation. I usually combine the treatment with rest in bed and restriction of food to 1000 c.c. of milk a day for a period of three to six days.

Let me now consider the value of this treatment in the second group of cases—the myocardial and arteriosclerotic. The prognosis in arteriosclerosis is not nearly so favorable as in valvular cases, especially if a complete breakdown has already taken place. The constant strain of the enormous peripheral resistance, and the direct interference with nutrition tend to a progressive diminution in the activity of the heart muscle. Thus, failure of compensation may appear with very little warning during the treatment, and there is none of the security felt in the valvular cases, where a break comes only after some unusual strain. When a break comes, the usual increase in dosage is demanded. On resuming the daily dose, however, we often find that 0.1 gram twice a day is needed. Soon even this will not postpone the day of reckoning, and larger doses are out of the question on account of cumulative phenomena.

This subject is well illustrated by the following history:

Case II.—Mr. B., aged fifty years, a schoolteacher. During the last four to five years he has had occasional attacks of shortness of breath, which has become almost permanent in the last four months, and is aggravated at times by a dry, hacking cough. Considerable œdema has appeared in the last four weeks. Examination showed chronic nephritis, hypertrophy and dilatation of the left ventricle, mitral and tricuspid insufficiency, œdema of the legs, and swelling of the liver. Blood pressure was 240 systolic, 160 diastolic (von Recklinghausen); pulse, 121, regular.

Compensation was secured in six days with restriction of food to 800 c.c. of milk, and the use of 0.8 gram of digitalis leaves. The pulse fell to 80, and the blood pressure to 170 systolic and 85 diastolic. He was dismissed from treatment May 15, but returned on June 13, with œdema of the legs; pulse, 104; blood pressure, 221 systolic, 165 diastolic. He was given 10 drops of digalen three times a day until July 10, when it was discontinued, as there was no change in his condition. Full digitalis action was then obtained with the powdered leaves, 0.3 gram on the first day, 0.2 gram on the second and third, and 0.1 gram on the succeeding days. This time he remained well and fairly compensated for nearly a year. On October 14 his pulse was 84; blood pressure, 210 systolic, 125 diastolic, and there was no œdema. About this time he had two short attacks, with anginoid symptoms.

On May 15 of the next year he returned with considerable œdema of the legs; pulse, 120, slightly irregular; blood pressure, 210 systolic, 160 diastolic; heart considerably dilated, and the liver 5 cm. below

the costal margin. He was given 0.8 gram of digitalis in the next four days, with the usual result of diuresis and disappearance of the œdema. After this it was found necessary to give 0.2 gram daily. This preserved a balance for about four weeks, when the œdema returned, and death followed in a few days.

In the second group of cases there is one other symptom to be watched for with the œdema, the enlargement of the liver, and the rales, and that is gallop rhythm. Lately, Müller[10] has again insisted on the importance of this as a sign of beginning failure, and an indication for the use of digitalis. I have followed this rule for several years with very satisfactory results. We practically never see this symptom with purely valvular lesions.

The following practical points should be observed in the use of digitalis in tonic doses:

Nothing is more important than that the digitalis should be physiologically active. Focke,[11] Gottlieb,[12] and Fraenkel[13] have repeatedly urged the necessity for standardized preparations, and anyone who has seen the absolute inertia of the usual infusions, tinctures, and digitalins will heartily agree with them. Only three preparations are available for tonic administration. Groedel, in his first publication, advocated digalen, which is supposed to be digitoxin, and without any cumulative action. Fraenkel showed that digalen has the same cumulative action as any other preparation if given in corresponding doses. I have discarded digalen, because it has often failed to produce any effect when afterward another preparation succeeded—just as I observed in the second case reported herewith. Balfour has used Nativelle's digitoxin granules with excellent results, and I also have found them very reliable. The dose is 0.00025 gram every day, and the action becomes marked after ten to fourteen days.

For some time now I have prescribed almost exclusively Allen's English digitalis leaves in doses of 0.1 gram a day, and concur with Janeway[14] in praising their uniform and reliable action. Secondary effects are rare, and I do not remember a single case in which the drug had to be stopped on account of stomach symptoms. In an experience of several years, I have seen but one instance of cumulative action with the daily dose of 0.1 gram. That occurred in Case I. herewith recorded, who developed the following symptoms after using the drug a year. There was a progressive slowing of the pulse from 80 to 68, and long silences of three to four seconds would be followed by a series of rapid contractions. The patient felt uneasy, was dizzy, and there was a considerable diminution in the

[10] Münch. med. Woch., 1908.
[11] Berlin. klin. Woch., 1906; Archiv der Pharmacie, 1903.
[12] Zur Theorie der Digitaliswirkung, Med. Klinik, 1906.
[13] Ergebnisse der innere Medicin, vol. i.
[14] Amer. Jour. Med. Sci., 1908, cxxxv, 781.

amount of urine. These symptoms disappeared upon stopping the digitalis. Such action can be avoided easily by giving the drug only three weeks out of four.

Probably the main reason why the tonic use of digitalis has not become popular is that men have feared that the body would accustom itself to the drug, so that the dose would have to be increased, and finally, there would be no action at all. Practical experience has shown that in purely valvular cases this does not occur; on the contrary, the dose may have to be reduced later, as described in the preceding paragraph. In the second group of cases, the dose had to be increased to 0.15 or 0.2 gram a day, but this was due to the progressive character of the process. Such an increase is shown graphically in the record of digitalis taken yearly by Kussmaul's patient.

Year.	Grams.
1892	15.00
1893	41.65
1894	35.10
1895	39.20
1896	44.48
1897	49.08
1898	57.86
To March 29, 1899	22.92

Experience has shown also that cumulative doses have exactly the same effect on patients who have been under tonic treatment as they have in similar cases in which digitalis is used for the first time.

We frequently have to consider the advisability of combining other drugs with digitalis. Many writers advise the giving of vasodilators to counteract the constrictor effect of digitalis. It is high time that we discard this hoary tradition and look squarely at the facts in the case. As mentioned at the beginning of this article, most of the work done on digitalis was with toxic doses, and vasoconstriction is one of the toxic effects. It has been shown clinically that in the majority of patients with beginning digitalis effect the blood pressure falls. Conclusive proof that digitalis in clinical doses relaxes the peripheral vessels has been given by O. Müller[15] and his pupils, who showed with the plethysmograph that there is a constant increase in the volume of blood in an extremity after the use of digitalis.

The increase in blood pressure so frequently seen in failing compensation, even in mitral disease, seems to be due largely to the dyspnœa. The giving of digitalis leads to better ventilation of the lungs; more oxygen for the respiratory centre, and secondarily a fall in blood pressure.

Regular weekly to monthly determinations in all my cases have shown that the tonic use of digitalis has no tendency to increase the

[15] Verhandlungen des Congresses f. innere Medicin, 1909.

blood pressure, but, on the contrary, a fall in the systolic pressure can often be noted coincident with an improvement in the general condition of the patient. Iodide has been used in conjunction with digitalis when it was deemed advisable. In anemic cases, when the general nutrition is badly affected, I have found the combination with arsenous acid very efficacious, and it may succeed when digitalis alone would fail.

Conclusions. 1. Digitalis acts as a specifie on the tonicity of the heart muscle, and is indicated whenever symptoms point to a failure of that function.

2. Its tonic effects are best secured with a dose equal to the amount excreted—about 0.1 gram a day—though doses of 0.15 and 0.2 gram a day sometimes can be taken for weeks without the appearance of cumulative effects.

3. Loss of tonicity is shown first by general symptoms—rales over the bases of the lungs, enlargement of the liver, and slight œdema of the ankles.

4. Digitalis is indicated, therefore, whenever these symptoms appear, and especially in cases in which the patient, after recovering from a severe break in compensation, shows a tendency to fail on the slightest exertion.

THE TREATMENT OF TYPHOID FEVER.[1]

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Etiology. Typhoid fever, which is one of the commonest diseases in our experience, but less common than in the experience of our medical fathers, thanks to the interest elicited by the work of the research scholar and an awakened sense of civic responsibility, is due to the invasion of the body by a definite bacterium, Bacillus typhosus of Eberth. The gross lesions are referable to the intestinal canal, but they are to be looked upon merely as local expressions of a general infection, just as lobar pneumonia is but a localization in the lung of an organism that infects the whole body. This is an important therapeutic concept, as it directs our attention to the individual as a whole and spares us useless effort to medicate the intestinal canal, in the hope of curing the disease. The mortality varies in different epidemics and varies in private and hospital practice, but on the whole is in the neighborhood of 15 per cent.

Therapy. We are dealing with a self-limited disease, the cure of which depends on the elaboration of specific bodies by the tissue cells of the patient, a result we have not yet been able to duplicate

[1] A lecture delivered at the Cornell University Medical College.

in the laboratory. This does not mean that we cannot influence the course of the disease. On the contrary, a considerable fall in mortality has come about as the result of skilled care, conscientious watchfulness, competent nursing, and the application of definite measures to the relief of symptoms and complications.

Rest. It would seem almost superfluous in a disease of such import as typhoid fever to insist on rest, but it must be remembered that the onset is slow, and in mild cases, or what at the beginning promises to be a mild case, the patient is prone to look upon it as an indisposition, and the physician may be uncertain of his diagnosis, and in the meantime the patient is on his feet, perhaps attempting to attend to his business, or at least protesting against the bed. These cases are the so-called "walking typhoids." Statistics have shown that these cases do not do as well as those receiving attention promptly, and it is probable that what might have been a mild typhoid attack has been converted, by lowering the resistance of the patient, into a severe or fatal case.

Where, then, suspicion is aroused as to the possibility of typhoid fever, the patient should not be permitted to waste his precious strength while one is awaiting the development of diagnostic symptoms or the result of blood examinations. He should be put to bed at once and prepared for what is likely to be a long siege. Real rest can be obtained only by careful and competent nursing, by the exclusion from the sickroom of all business cares and causes for anxiety, and refusal to make the sickroom a reception room for solicitous friends.

The patient is not to leave the bed to go to the toilet, or even to use a commode, but the bed-pan is to be insisted on. This trouble comes in the early days of the fever. It will be a source of argumentation and contention, but one's dictum must be firm, backed up by a few words of explanation. The need for rest, the need of sparing the tissues useless oxidation, is emphasized by the length of the disease.

Bed. While remembering to conserve the strength of the patient, it must not be forgotten that the strength of the attendant ought to be economized, and in no way can this be done to better advantage than in selecting a bed on which the patient can be readily handled. The ideal bed is the half-bed, or single bed, of a convenient height, with strong woven wire springs and a firm springy mattress. A folded blanket will render the surface smooth. Over the blanket, is to be placed a sheet, a rubber sheet, and a draw sheet. Sheets should be drawn smooth, for freedom from wrinkles and dryness and cleanliness are great factors in preventing discomfort, and, in very sick patients, the formation of bedsores. Over the patient a sheet and a light blanket is sufficient; and one low pillow, not too soft, makes for comfort. The upper sheet should not be drawn too tightly over the feet of the patient, as is often done to improve the appearance of the bed, to the patient's great discomfort.

Room. The best room is the largest room, with the amount of light most gratifying to the patient; with sunshine in the colder months, and shade in the summer months. It should be well ventilated and free from encumbrances. A hardwood floor is best. If there are carpets, they should be protected in the vicinity of the bed, in consideration of contamination with secretions, which may occur in the ordinary handling of the patient. It is very desirable to have a bathroom near at hand, for the convenience of disinfecting and disposing of secretions, as well as for facilitating hydrotherapeutic measures.

If there is a balcony or piazza that may be approached from the room, it meets a great desideratum, for in summer the patient ought to be kept out of doors, and in winter I am convinced of the value of cold air in improving the nervous manifestations and general condition of the patients. I believe we shall use the open-air treatment for all infectious fevers with vast benefit.

The bed should be prepared by placing a blanket on the wire springs, and on this a rubber sheet, each extending well below and on either side of the bed. On these is placed the mattress, on which the bed is made as usual. Over the mattress and its coverings the blanket and rubber sheet are now folded like an envelope, thus preventing the entrance of air under the clothes. A hood is worn by the patient and a hot-water bottle placed at the feet; the nurses are dressed for out of doors.

When there is diarrhea and incontinence of the bowel or bladder, then more constant attention to the bed does not make the open-air treatment feasible. If the patient is very sick or is delirious, a nurse should be in the room day and night, as self-destruction has occurred in this condition.

Care of the Body. If a properly trained nurse is in attendance, she will exercise all those niceties of her profession with reference to the care of the patient's body and comfort, which will usually improve on one's own suggestions, but it is unjust to the patient to take that for granted, and a quiet survey of the situation and inquiry into the methods used is proper. However, all nurses are not properly trained, and all patients cannot afford the attendance of a nurse. Under these circumstances, one must know what should be done; one should write out explicitly what is to be done, and then see that the instructions are comprehended and successfully carried out.

The state of the patient's mouth requires constant attention, lest, from a foul condition, it become the source of infection of the mucous membranes and their lymphatic supply, the tonsils, the ears, and the lungs; and, upsetting the stomach, destroys the appetite. The teeth should be brushed two or three times a day. The mouth should be rinsed with water or boric acid solution (2 per cent. to 4 per cent.), or one of many mild alkaline and antiseptic washes, after each feeding.

If there is a heavy coat on the tongue, such may be removed by careful scraping with the edge of a whalebone. Especial care must be taken to get at the back of the tongue and remove thick mucus from the throat. The cotton swab on a toothpick is a ready means of getting at the dead spaces in the mouth, where food may collect and decompose.

If there is much sordes, and there are fissures, a mild antiseptic containing phenol is of value. Here is one recommended for the purpose:

℞—Phenol solution, 1 to 20,
Glycerin . . . . . ää 1 ounce
Boric acid, saturated solution . . . . . 8 ounces—M.
S.—To be used as a mouth wash.

Bits of cracked ice are grateful, and the conditin is much relieved if the patient gets enough water to drink. The nose is to be kept clear by the use of olive oil on a swab, to soften dried masses, and cleansing with boric acid solution or mild alkalines. Whether hydrotherapeutic measures are pursued or not, the body is to have a cleansing bath with warm water and soap and the sponge.

The formation of bedsores is the *bête noire* of a good nurse, and this she avoids by constant care of the skin. Moisture about the buttocks is quickly removed, the parts sponged with alcohol or alcohol and water, equal parts, and, if the buttocks have been much soiled, with a mild antiseptic solution—phenol, 1 to 40, for example. If pressure has been long continued, the skin is rubbed to improve the circulation in the part. The parts are then dusted abundantly with a drying powder, like any of the talcum powders used for toilet purposes. The skin of the whole back is treated in this way. Especial attention is paid to the sacrum, the anus, the buttocks, and the heels. The position of the patient should be frequently changed, not only to avoid bedsores, but to lessen the chance of hypostasis in the lungs.

If sores are threatening, rings are used to take the pressure off, but unless used intelligently, they may be more annoying than useful. If bedsores develop, it may be imperative to use a water-bed or air-bed. The excoriated skin may be covered with a drying powder, like aristol, or a bland ointment, like zinc oxide ointment. If more serious sores appear, they are to be treated on surgical principles.

At certain times and in certain places flies are a great annoyance to the patient, and, not only that, but a very real danger to other members of the family or vicinity, by their well-known ability to carry infection from the patient to the food or drink of those about him. Under such circumstances, screens in the room and over the patient are all-important.

This leads naturally to a consideration of the disposition of the secretions and disinfection of the patient's clothes and utensils.

DISINFECTION. The stools, the urine, the clothes, the utensils, the bath water, the sputum, and the vomitus have all to be taken into consideration. Stools may be disinfected by 1 to 20 phenol. At least twice the volume of the stool should be used, the stool well broken up and allowed to stand several hours. Chlorinated lime, too, is used. It must be fresh, as it soon deteriorates on exposure to the air. A 1 per cent. solution is required; or, to avoid making exact solutions, a handful is put in the pan with the stool and enough water to cover and mix it with. Bichloride is not reliable for disinfecting stools. Urine is disinfected with 1 to 20 phenol, using one-third to equal amounts. The lesser amount has been shown to be effectual after one and one-half hours' contact. 1 to 1000 bichloride, in volumes equal to $\frac{1}{15}$ to $\frac{1}{40}$ of the urine, standing for an hour and a half, is effectual.

Bath water may be disinfected by adding $\frac{1}{2}$ pound of chlorinated lime to a tub (200 liters) of water and allowing it to stand an hour.

Bed linen should be soaked in 1 to 20 phenol for two hours or in formalin, 3 ounces to a gallon, for twelve hours, and then thoroughly boiled. Bed-pans, urinals, rectal tubes, and rubber sheets can be disinfected in phenol 1 to 20 and allowed to soak in it.

Knives, forks, spoons, and crockery can be thoroughly boiled. Sputum is best burned; vomitus may be treated with phenol and lime.

Mattresses can be disinfected after the disease is over with dry heat, and the room should be disinfected.

The nurses should give tub-baths, wearing rubber gloves and a rubber apron. If given with the bare hands, and, indeed, after handling secretions, the hands should be carefully washed and soaked in 1 to 1000 bichloride.

It must be remembered that the administration of urotropin by the mouth disinfects the urine, but we should not intermit the precautions just named.

DIET. Everyone who has treated typhoid fever has felt that the most urgent problem he has to meet is the dietary. He has consulted authority with the result that he has come away more bewildered than instructed. He has been urged to maintain a low diet, lest too great a burden be thrust on digestive organs and tissues weakened by disease, or damage be done to the inflamed intestinal wall with the production of hemorrhage or perforation; on the other hand, he has been taught to give the patient great freedom in diet, lest strength be sacrificed as the result of starvation. He has read arguments for liquid food, for solid food, for stimulating food, for bland food, until in his despair he chooses some guide with whom he is more or less well acquainted, copies his directions with slavish exactness, and proceeds to feed all patients by this measure.

We are dealing here with a fever and a long fever, during which oxidative changes must be met, either at the expense of food ingested or of the body itself, and during which extensive tissue destruction

is going on as the result of the disease; this must be made good by the food or the deficit will increase continuously up to convalescence, or bring about a bankruptcy, before convalescence can be attained.

The first great fact to be kept in mind in all fevers is that the body demands as much energy and heat in terms of heat units in fever as it does in ordinary rest in health; we will say 33 calories per kilo, or some 2300 calories for a man of 70 kilos, or 154 pounds; and that as a result of the fever itself, he usually needs some 25 per cent. more, or about 40 calories per kilo, or some 2800 calories for a 154-pound man. Therefore, he ought to get 2800 calories to meet this need from his food, unless he takes it out of his own body.

The second great fact with reference to the body needs to be met by the food, is that there must be a certain minimum of protein in the diet. Voit's standard was something over 100 grams per day, but more recent work has shown that even less would do, but that some 70 grams should be afforded. This demands intelligent scrutiny of the food, with the knowledge of its composition, in order that this minimum should be observed; or, what is next in importance, that it should not be too much overstepped.

The third great body of facts, in dealing with diet in fever, are those which inform us of the loss of protein to the body in fever and how to shelter it by the food administered. The wasting after fever, even a short fever, and so emphasized after a long one, is one of the most familiar phenomena of disease. The wastage is, of course, due to loss of both fat and protein, but more especially, from the standpoint of importance, of protein.

The destruction in fever may be assigned to three particular causes: (1) Starvation. There can be no doubt that our patients are, in too many instances, underfed. That this cause for protein loss can in a goodly measure be prevented goes without saying. (2) Fever itself. Pyrexia exerts a destructive action on protein of the tissues, as can be determined by studying the nitrogenous output in fever induced artificially, as in hot baths or by puncture of the floor of the fourth ventricle. In disease, however, it is not always easy to separate it from: (3) toxic destruction, meaning the breakdown induced by the toxins of the infecting organisms, in part autolytic, perchance, but the detail of which is still obscure.

The influences of pyrexia can, to a certain degree, of course, be influenced by hydrotherapeutic and other antipyretic measures. The effect of the toxins, until we have specific sera or other specific measures at our command, however, are not under our control; and yet, it is the purpose of this thesis to show how near to a nitrogenous equilibrium a patient can be brought, in the face of this toxemia, if supplied with the proper food in the proper manner.

I am indebted to Dr. Shaffer and Dr. Coleman[1] for many state-

[1] Archives of Internal Medicine, vol. iv; Jour. Amer. Med. Assoc., 1908, li.

ments of facts made here, but they are not to be made responsible for any conclusions I may draw.

In the disease in question wasting is marked. A loss of 10 pounds is slight, but losses of 40, 50, 60 pounds or more may occur. In this loss, nitrogenous tissue takes a large share. Cases in which the nitrogen loss has been studied, show the equivalent of 7 pounds of pure muscle tissue in twelve days, of 5½ pounds of muscle tissue in eight days, and not rarely 1½ pounds of muscle tissue in a day. This loss may mean not only a deprivation to the body of the functions this tissue subserve, but also disturbances, perhaps toxic in character, induced by the effort to katabolize this protein. The nitrogen partition offers similarities to those seen in the toxemias of pregnancy, and, to quote Dr. Shaffer's words, "there are severe so-called toxic cases of typhoid fever which terminate with acute yellow atrophy of the liver, a condition which appears to be closely associated with a particular type of faulty protein metabolism."

This loss can be avoided, to a greater or less degree, by the food. In considering the foodstuffs, fat, carbohydrates, and protein, one would naturally turn to the proteins to make good a loss of proteins, and, indeed, that is necessary, but to no such degree as would be anticipated. There must be enough protein to meet the daily needs under ordinary circumstances—the 70 grams of which I spoke. There may be a slight storage of excess, which does not obtain in health, but if we give a frank excess, it must be destroyed and eliminated by the organism, and more than that in the process of its katabolism, by virtue of what is known technically as the "specific dynamic action," gives rise to a large amount of heat, which cannot be utilized for the purposes of the body, and extra burden is placed to dissipate the heat. The result is that about 140 calories of protein are required to secure 100 calories available for the purposes of the body.

Fat, on account of its well-known tendency to induce digestive disturbances and diarrhœa, must be used sparingly. According to the best known investigators in this field, the strongest sparers of body protein are the carbohydrates, and it is upon these that we place our reliance to solve the problem. It has been shown that the absorption of the fats, carbohydrates, and protein in typhoid fever, when severe diarrhœa is not present, is within 10 to 15 per cent. of normal, and Folin has shown that the absorption of the carbohydrates in this disease is practically normal.

In the feeding experiments carried out along this line of reasoning, among the typhoid fever cases in the wards of Bellevue Hospital the carbohydrate selected was milk sugar, because it ferments with difficulty and is less sweet to the taste than cane sugar and more soluble than the starches. The experiments showed that a nitrogen gain could be attained when the protein intake amounted to 75 to 85 grams, while at the same time the caloric content of the food was

high, 3700 to 3900, or 70 to 73 calories per kilo; that is, enough protein was used to meet the daily needs and the loss of nitrogen was stayed by the high carbohydrate content of the food. When the same amount of protein was used with 32 calories per kilo, the loss was considerable. When only 16 grams of protein were used, but with 62 calories per kilo, the loss was greatest.

Now, in one of our most recent and authoritative systems of medicine, the diet advised is 4 to 6 ounces of milk every four hours, or 24 to 36 ounces a day; the whites of one or two eggs every four hours, making the whites of six to twelve eggs a day. Taking the larger amounts, and granting that the patients were fed night and day, we should have 730 calories from the milk, and, allowing 60 grams as the weight of an average egg, two-thirds of which is white, we should have in twelve eggs 480 grams. Of the white, 12.5 per cent., or one-eighth, is protein; the fat and carbohydrate almost negligible. That would give 60 grams of protein from the eggs, or 246 calories. These patients would get at the most 1450 calories, or about one-half their theoretical requirement. Of protein, they would get about 100 grams, a sufficiency, if backed by enough carbohydrate, This diet has been shown, in the Bellevue patients, to be accompanied by a large negative nitrogen balance. There may be theoretical considerations for such a low diet in typhoid fever, but I do not think that they have any scientific basis.

The more liberal feeding which has always been insisted on by individual clinicians, who, however, formed a small minority, is in the hospitals of New York today finding more advocates. The dietary at St. Luke's Hospital, contains on an average 63 ounces of milk to which 1 ounce of milk sugar is added, and three whole eggs a day. This diet contains 2585 calories, about 1000 less than Shaffer's and Coleman's and about 85 grams of protein.

The basis of the high caloric diet as used in the wards of Bellevue Hospital is milk, cream, eggs, milk sugar, bread, and butter. A quart of milk affords 650 calories. A quart of cream (20 per cent.) about 2000 calories; each egg 60 to 70 calories; milk sugar about 120 calories to the ounce; white bread, home-made, about 1225 to the pound—that is 100 calories to a thick slice of $1\frac{1}{4}$ ounces; butter about 3600 calories to the pound, or 100 calories in a pat a trifle under $\frac{1}{2}$ ounce. With such materials various combinations may be made: $1\frac{1}{2}$ quarts of milk will give 1000 calories, a pint of cream 1000, $\frac{1}{2}$ pound of sugar about 1000 more, four eggs 250 more, thus making 3250 calories. If one wants a few calories more, 250 to 500 more can be gotten in 2 to 5 ounces of sugar added to lemonade..

A glass of milk that contains 7 ounces of milk, 1 ounce of cream, and 1 ounce of milk sugar, offers about 325 calories. Eight such glasses in twenty-four hours will give 2600 calories, as high as many care to go. The protein, as well as the calories, in this food must be remembered. The milk contains 35 grams to the quart, and

the cream a trifle less, 25 grams; the eggs, 8 or 9 grams; the bread, about 9 per cent., or 45 grams to the pound loaf. In the more liberal dietary just suggested, about 100 grams are afforded; in the smaller, 70. Two eggs added to this would increase the protein to 85 grams.

Dr. Shattuck, of the Massachusetts General Hospital, has long advocated a more liberal dietary in typhoid fever, and can point to quite as good results as the advocates of a restricted or milk diet. I will not cite his dietary in full, but will mention some of its items, which show one how varied we can make our patient's food and relieve a monotony that threatens a loss of appetite and gastro-intestinal disturbances. These are milk and the various milk products, some of which are better borne than others, such as buttermilk, koumys, matzoon, whey or milk with tea, coffee, or cocoa; or the milk can be diluted with lime water, Apollinaris, or Vichy; soups of beef, veal, chicken, tomato, potato, pea, bean, or squash, strained and thickened with powdered rice, arrowroot, flour, barley, or egg; gruels, ice-cream; eggs, soft boiled or raw, or eggnog; finely minced lean meat or scraped beef, soft crackers with milk or soups, soft puddings, soft toast without crusts, blanc mange, wine jelly, apple sauce, and macaroni.

Many of these articles have a low calorie content, and would not serve the purposes of our new teaching, but they show what is safe and what may be used to diversify the dietary.

Each patient must be carefully studied with reference to his dietary. We can make a mathematical problem of his food needs, but not of him. If he is highly toxic and stuporous, if he has gastro-intestinal disturbances, we cannot expect him to ingest or digest the same quantity and quality of food that he would if his disease was of a milder type. We wish to avoid the older policy of frankly starving our patients, while we at the same time do not desire to stuff them like a Christmas goose, willy-nilly. I believe if the patient is moderately or severely ill when he comes under our observation, we should begin on milk for the first twenty-four hours, some two quarts; then a little milk sugar, a dram to a glass, and rapidly more as he shows that he handles it well; and at the same time or shortly after, a little cream—a half ounce to an ounce to a glass, watching this with especial care; eggs, gruels, or soups, bread or toast or crackers, ice cream, cup-custard, or other milk modifications. If the milk is not well borne, milk preparations, such as koumys, matzoon, buttermilk, or whey, may be used.

If the tongue is coated, if there are eructations, or if diarrhœa sets in, the cream should be stopped and the milk skimmed. Of the meat soups, one may say that they have little caloric value, but may improve the appetite. The scraped or strained meats may be allowed, if greatly desired, but they are not an economical form of food in fever, owing to their high specific dynamic action, and should

not be given in the severe toxic cases, as it is believed, on fair grounds for such an assumption, that the liver in some of the cases is impaired and unable to metabolize the protein of the food brought to it in the normal manner.

At any rate, in such cases the urea nitrogen decreases while the "rest nitrogen" increases, and the giving of meat aggravates the condition and furnishes us in the convalescence of severe cases the so-called "febris carnis," as Ewing and Wolff believe.

I believe a patient should be given as much food as he will take and handle well. If he will take 4000 calories, he should have it; but with the earliest signs of gastrointestinal disturbance or disgust for food, one should diminish the food, and do so promptly. The patient's appetite is a valuable guide, and within the range of food mentioned, his likes and dislikes should be considered.

HYDROTHERAPY. If one had a brief to hold for hydrotherapy, he could not do better than to appeal to its application in typhoid fever. So thoroughly convinced is the American practitioner of its great value in this condition that arguments to maintain this thesis are only of historical value. The mortality of typhoid fever, vary as it may in different epidemics, hospitals, and localities, has, under the old expectant treatment, amounted to 15 per cent. the world over. Since the introduction of the cold water treatment, where large numbers of cases can be collected, as in great hospital services, the mortality has been cut in half, running about 7.5 per cent. with wonderful uniformity in the hospitals of our larger cities. Statistics have no value unless applied to large numbers. For example, in a certain hospital some 100 cases of typhoid were treated by the bath, with a mortality of 1 per cent. One might have attributed this success to some perfection of technique not prevailing elsewhere, but when the same clinician had collected 400 cases his published mortality showed 7.8 per cent., or that of the general experience.

The hydrotherapy of typhoid fever, as universally adopted in this country, is based on the Brand bath, or a modification of it, and the greater the modification, the less successful it seems to be. Two emphatic statements with reference to this bath should be made: (1) The essence of the bath lies in the application of cold water and friction, neither one nor the other alone, but both; (2) the purpose of the bath is to improve the condition of the poisoned centres and organs: the heart, lungs, kidneys, and centres in the brain and medulla; incidentally, and to a degree beneficially, to reduce temperature. The last is too often put first, which in this instance does not have biblical justification.

METHOD. The body should be immersed to the neck; the bath should be given preferably in a tub. In hospital practice, a portable tub on wheels is used. In private practice this is, as a rule, too cumbersome, and a tin bath tub may be used, elevated to nearly the

level of the bed on wooden horses, blocks, or other contrivance. Placed alongside of the bed, the floor being protected by a rubber sheet, oilcloth, or carpet, it is filled three-quarters full of water, with buckets, or a hose attached to a tap, if one be conveniently near.

The patient's nightshirt is removed, his genitals covered with a napkin, bound about the body, or the whole body covered by a sheet, under which he is bathed, which latter method is a little more awkward. He is given a half ounce of whisky, or a cup of hot strong coffee (4 ounces), the face bathed with cold water, a folded bandage a couple of inches wide, bound around the forehead and tied below the occiput, to keep the water applied to the head from running into the eyes and streaming down the face, and he is then ready to be lifted into the bath. To do this, the best way is for one attendant to rest the patient's head on one arm while he raises the upper part of the body by lifting under the shoulders, while another attendant lifts the lower extremities, the patient being requested to stiffen himself out.

Another method, which has advantages if the patient is very stuporous, is to spread a hammock netting under him or a strip of canvas with straps attached, and lift him on this into the tub and by the same means out of it. The head should rest on a water-cushion ring or air-ring.

As soon as the patient is immersed in the bath, the equally important part of the procedure is begun—the friction, gently applied (not kneading) to all parts of the body except the abdomen. The back must not be forgotten. At intervals during the bath, cold water, at 50° F., should be poured over the head. While in the bath the bed should be prepared for his reception, by placing on it a double blanket, on the side he will occupy; a pillow is covered with a towel, the blanket is covered with a linen sheet, and hot-water bottles are placed at the foot.

The patient is then lifted onto the bed, the napkin is removed, and he is surrounded by the sheet, the edges and ends being tucked about the neck and under the arms. He may be allowed to lie in the sheet for five or ten minutes and then dried with soft towels, or more immediately if the temperature is low and he is shivering. The tub is best emptied by a siphon.

The temperature of the water Brand set at not more than 70° F., and not less than 65° F. The frequency he advised was every three hours, if the rectal temperature was above 102.5° F.; the length of the bath, fifteen minutes. These rules laid down by Brand have met with but slight modifications where satisfactory results are obtained. Slightly lesser temperature of water is used at times, a little higher temperature of the body taken as the indication of the bath, and the initial bath begun at a milder temperature. At St. Luke's Hospital, in New York, the tub bath is given at 80° F., and in selected cases as low as 70° F. The bath is con-

tinued fifteen minutes. It is given every four hours, if the rectal temperature is 103° F. At the Presbyterian Hospital, in New York, the tub bath is given at 85° F. for ten to fifteen minutes, at four-hour intervals, if the body temperature is 102.5° F.

It is fully appreciated by the advocates of the Brand system that the reaction to a bath at 65° F. cannot be anticipated in markedly toxemic cases, so that a patient seen in the third week or latter part of the second, who is very toxic, should be subjected to a bath of higher temperature (75° F. or 80° F), and for a shorter time. Our hospitals rarely get patients in the first week, and the higher temperatures adopted are better suited to their class of cases.

If a tub bath cannot be obtained, a substitute for one can be constructed in the bed by running a clothesline around the bed, at a suitable height above its level, attaching a rubber sheet to it by clothes-pins, thus making a tub of the sheet. Other contrivances can be resorted to, to approximate a tub in the bed, as building around the sides and foot and head with rolled blankets, and the use of the rubber sheet as the receptacle for the water. When the patient is put in the water one expects a sudden shock, deep breath, and gasping. These are in themselves beneficial. The patient may shiver, the skin may be shrivelled, and the nails cyanotic, and yet not contraindicate the bath; but if the face is cyanotic, shivering marked, and chattering of the teeth occur, and the patient does not react well on being taken from the bath, then the next bath should be at a higher temperature and less long. Cyanosis of the face or threatened collapse should lead to immediate removal of the patient, warmth to the body externally and internally, and stimulants—hot coffee or hot whisky.

Many untoward symptoms in the bath are due to failure to appreciate the value of, and failure to apply properly the friction during the bath.

The benefit derived from the bath is very obvious and is expressed by improvement in the functions of various organs. No benefit can be expected from any procedure applied to the moribund, and the greatest success in the treatment of typhoid fever by this method is in the early application of it. Many of the severe symptoms seen in cases coming under observation or treatment late, are not observed at all in the cases bathed the first week. The most characteristic effect clinically of typhoid fever is the toxemia impinging on the brain, inducing restlessness, sleeplessness, delirium, stupor, and subsultus tendinum. The stimulating effect on these centres, receiving sensations pouring in from every point of the periphery and nourished by a better blood supply, is shown by an amelioration of every one of these symptoms, better sleep and more quiet, and a clearer mind more alive to its environments.

In typhoid fever the pulse is slow; when rapid, ominous; it is often dichrotic, bespeaking a low vasomotor tone. We stand in

dread of heart failure, but the opinion prevails today that in acute infectious diseases the heart rarely fails, but that the vasomotor apparatus is the first to falter.

After the tub, the pulse is smaller and harder, losing its dichrotism; the pressure increases by 15 to 20 mm. Hg., and the rate falls. All this is very real, and may be attributed, in all probabilly, to the effect exerted on the vasomotor centres, though the tonic effect on the vascular walls of the periphery is undoubtedly a factor.

On the respiratory apparatus a beneficial effect is exerted by the fact that the contact with the cold water causes the patient to take deep breaths, which lessen the danger of passive congestion and hypostatic pneumonia that threaten in severe cases. More than that, the respiration of the tissues is enhanced, as increased oxidation can be demonstrated by measuring the oxygen intake and the CO_2 output.

The contact of the cold water and the friction improve the circulation in the skin and so improve its functions. The improved condition of the circulation increases the elimination of urine and the toxins of the disease. The temperature is lowered, but this is no longer considered the object of the treatment, and often in the height of the disease the temperature is not influenced, but striking improvement in the general condition is attained, in terms just given.

After all, it is the individual that is being treated, and this means that a precise procedure cannot be the optimum for every case. Higher temperature of the water and shorter baths are required for those who stand the shock of the typical bath poorly, for those toxic and advanced in the course of the disease, and for children.

Contraindications for the baths comprise hemorrhages and signs attributable to perforation or peritonitis, all of which conditions demand absolute rest until the proper procedure, surgical or otherwise, can be carried out. Cholecystitis, phlebitis, pleurisy, if accompanied by considerable pain, contraindicate the bath for a like reason. Cyanosis and syncope demand cessation of the bath in which they occur, but if the case is a severe one, the use of water may be continued in a milder form of application. Large bedsores, because of the difficulty of properly treating them, contraindicate the bath. Bronchitis, pneumonia, the milder grades of kidney involvement, and menstruation and pregnancy do not call for a change of procedure.

Beside the full bath of Brand, there are certain other methods of applying water in this disease. A slight modification of the Brand bath is the so-called graduated bath of Ziemssen. The details are the same as for the Brand bath, except that the water is warmed to 90° F., and then, after the patient is in the bath, is cooled down to 70° F. by adding cold water as the bath proceeds. The bath lasts half an hour with friction. This is a suitable procedure for those who stand the bath poorly, or think they do, and refuse the colder bath, or the very toxic cases of the later stages, or children.

When, for any reason, it is not feasible to use the tub, an approximation to the methods and results of a tub are attained by what is sometimes called a slush. The patient is stripped, a rubber sheet of ample proportions is put under him, the edges of which are elevated by pillows and blankets folded to the formation of a trough. Water is poured into this trough and the patient given a bath with friction, as in a tub, while cold compresses are kept at his head. The water, of course, becomes warm rapidly and is kept cold by adding bits of ice. The temperature cannot be so accurately regulated as in the tub. The head of the bed is elevated, the water drained off, the patient rubbed with a little alcohol, or alcohol and water, after drying and removing the rubber sheet, and then dusted with talcum. These slushes may be very effectual when properly given.

When the baths are refused, or for other reasons cannot be given, packs and sponges may be substituted; but, while these afford comfort and do some good, they fail in the general stimulating effect that comes from the friction of the body immersed in cold water.

Care of the Alimentary Tract. In the routine treatment of typhoid fever there are no organs whose functions have to be so jealously guarded as those of the alimentary tract, not only because failure of digestion and absorption threaten a fatal issue in so long a disease, but because insufficient elimination and excretion heighten the toxemia, and because diarrhœa exhausts and hemorrhage and perforation await upon the advance of the local lesions. Constipation is almost constant in typhoid fever as we see it in New York. Diarrhœa, which one might gather from any of the text-books as characteristic, is rather the exception.

If seen early, a catharsis of castor oil, 1 ounce, or Epsom salt, 1 ounce, may be given. If seen late, all catharsis should be avoided. Enemas of tepid water, of soapsuds, or, in obstinate cases, soapsuds with an ounce of sweet oil, or castor oil, should be given every other day. It is the lower bowel one wishes to empty, not the upper, and this can be done effectually with the enema. The upper bowel, the site of the lesion, like any other inflamed part, should be given as much rest as is compatible with its physiological functioning.

The lower bowel is physiologically differentiated; the lower part forming the reservoir for undigested food, and, what is more important, the excreted matter, the nitrogenous content of which forms an almost constant portion of the total nitrogen output; it is, too, the site of active bacterial action, and is an absorbing surface. We wish to avoid absorption of toxic material of bacterial action, which may add its burden to the already overloaded blood. Nothing is more surprising than the large stools obtained from patients on a milk diet, and still more, the considerable movements obtained when patients are taking little or no food. The patient's plea to

avoid the enema that he has taken nothing, therefore, can have nothing to evacuate, must be met by explanation of the physiological fact and demonstration of its accuracy.

Tympanites, to some degree, is almost constant, but when marked is usually the expression of the severity of the intoxication. This paretic condition of the bowel, which allows the collection of large quantities of gas, is rife with danger, as it increases the chances of hemorrhage and perforation, and interferes with respiration and cardiac action; pain may be an accompaniment. The free use of water, the attention to periodical evacuation of the bowel, and the general tonic effect of the baths, lessen the severity of this complication. When, in spite of such precautions, it does obtain, the following measures may be carried out:

1. Introduce for a distance of 12 to 15 inches into the bowel a soft, pliable rubber catheter, taking great care not to use force. The larger bowel is, as a rule, more implicated than the smaller. This will at times permit the escape of considerable amounts of gas. It should be left in ten to twenty minutes, or even a half hour, and may be repeated every two to four hours, as indicated. Turning the patient while the tube is in place may facilitate the escape of gas.

2. The use of stupes. Two or three thicknesses of flannel are wrung out of hot water, as dry as possible, and applied every three or four hours. The technique is as follows: A flannel roller is put under the patient, the abdominal wall smeared with vaseline, the stupes applied, the edges being turned under to prevent dripping; oil silk applied, and the roller brought up over the stupes in the manner of a binder. This should be changed every two or three minutes for ten or fifteen minutes, or every ten or fifteen minutes, for several hours.

3. Turpentine. There are three ways of administering turpentine to relieve this condition: First, in the stupes, by adding a dram of turpentine to the hot water into which the flannels are to be dipped; by sprinkling the stupes with turpentine; or by very lightly passing a bit of absorbent cotton wet with turpentine over the abdomen before applying the stupes. Care must be constantly exercised not to irritate or burn the skin. Careless application of the turpentine will certainly entail such trouble. The second method is by giving turpentine enemas. One-half ounce to an ounce of turpentine is to be used in one pint of soapsuds enema. The method is as follows: Take a little hot water and a piece of Castile soap; make a thick soapsuds; add the turpentine slowly, constantly stirring. This makes an emulsion. Add the pint of water, and the emulsion remains stable. Oil enemas are made in the same way. The third method is by the mouth; 10 to 20 drops are used every four hours. It is sometimes dropped on loaf sugar, but the turpentine makes the sugar tough and not easily soluble. It is better given in capsules, 10 minims each, or in emulsion, to

which a little oil of cinnamon may be added as a flavor, while at the same time it adds to the effect of the turpentine. Thus:

℞—Olei terebinthinæ rectificati 10
Olei cinnamomi . . . . . q. s. (or 3)
Acaciæ . . . . . q. s.
Aq. destillatæ . . . . . q. s.
M. et fiat emulsum 60
Sig.—One or two teaspoonfuls.

Sometimes a large flat ice-bag or the ice-coil, brings relief, and if all these measures fail, a hypodermic injection of eserine salicylate or sulphate ($\frac{1}{50}$ grain) may be tried. The diet should be materially decreased, milk stopped, and albumin water administered, and water given freely, if the condition is severe.

Diarrhœa, fortunately, is not a common complication in typhoid as we see it here. Two or three loose movements a day do not call for interference, except in the matter of regulation of food. The food and neglect of the bowel may be the inciting causes. If the patient is on a milk diet, the fat is more likely to be responsible for the diarrhœa than any other constituent of the food, and should be removed. Scalding the milk may help, If the diarrhœa is severe, the food should be cut down. Of the astringents, bismuth is the best, and should be given in sufficient doses (30 grains) every two or three hours. This must not be kept up too long, or if administered over a long period, it must be remembered that the bismuth may be retained in the bowel in such quantities as to produce irritation by sheer weight, while the diarrhœa still continues; hence, the necessity for irrigations.

Prolonged saline irrigations, by improving diuresis, lessening toxemia, and keeping the bowel clear, may be of distinct advantage. It is tempting to use opium, but it cannot be too emphatically insisted upon that by so doing, pain, the earliest and most important symptom of perforation, may be lacking—to the patient's undoing. It should not be used unless exhaustion from diarrhœa and increasing danger of hemorrhage and perforation from peristaltic unrest seem to demand it. Then one may give a few large doses, rather than long-continued small ones; for example, a pill of opium ($\frac{1}{2}$ grain) every two hours for three or four doses, or some such marked astringent mixture as one of our well-known clinicians has advised:

℞—Pulv. opii . . . . . 0.25 (gr. iii$\frac{3}{4}$)
Pulv. camphoræ . . . . . 1.00 (gr. xv)
Plumbi acetatis . . . . . 3.00 (gr. xlv)
Bismuth. subnitrat. . . . . 30.00 (oz. j)
M. et div. in chart. no. xv.
Sig.—One every four hours.

Gastric distress and vomiting are not common. If vomiting does occur, the milk should be skimmed; or, if severe, stopped. Cracked ice, in small pieces, sucked, may be of avail. Then try a mild

mustard paste to the epigastrium, one part in four of flour. If these do not succeed, try a bland powder of cerium oxalate (3 grains), sodium bicarbonate (5 grains), and bismuth subnitrate (10 grains), every two hours. If very severe, cocaine hydrochloride ($\frac{1}{3}$ grain) in tablet form, or one teaspoonful of 0.1 per cent. solution every two or three hours. Finally, stomach washing may succeed when other measures have failed.

Few things are more trying than meeting the danger arising from hemorrhage and perforation. Few occurrences demand more judgment and common sense than treating the hemorrhage of typhoid. To judge by the advice given by some authors, the appearance of hemorrhage is to be treated like an invading foe, to be met with every weapon at our command, from stiletto to bludgeon; while more timid counsellors urge letting it severely alone, lest by interference we come off worse than before. In the first place, in the early stages, a little blood may appear in the stools, resulting from an oozing of the hyperemic patches, and such small amounts at such a period need no treatment. In the latter part of the second week, and during the third, we are anticipating the hemorrhages of real significance from the ulcers and know that they do occur in about 7 per cent. of the cases. If the hemorrhage is mild, that is, small in amount, or well borne by the patient, we may have no premonition of it until the stool shows the red blood of the recent bleeding, or the tarry stool of one occurring some hours before.

In these cases, the present bleeding is of less importance than that which may occur later, so that our treatment is largely prophylactic. We cut down the food, we enjoin absolute muscular rest on the part of the patient, and turn him gently when required.

We may apply a light flat ice-bag or coil to the abdomen. We may give calcium lactate in doses of 10 grains three times a day, and in the light of our recent knowledge of the effect of horse serum on coagulation, we may give a dose of 10 to 20 c.c. of it; or, failing that, diphtheria antitoxin in similar amounts. To get the best results from the serum, it should be as fresh as possible. The old serum loses much of its potency in this respect. If fresh horse serum is not available, fresh rabbit's serum may be used.

Antitoxin, because it is likely to be old, is less valuable, and when a concentrated antitoxin, made from the globulins, of no use at all. The same precaution must be used in administering the serum for this purpose as when giving antidiphtheritic or antitetanus serum, to avoid the results of anaphylaxis. To prevent this, a dose of 10 minims is given, which will be enough to produce sufficient evidences of anaphylaxis if it exists, but not a fatal result. If no manifestation appear in a half hour, the full dose may be given with more assurance. An initial dose of serum after a matter of ten days is likely to establish an anaphylaxis, and this precaution is doubly necessary, when a second dose is given after such an interval. Immunity

against anaphylaxis may be established by giving doses of serum—full doses—every day, or second day.

If the 10 minims cause anaphylactic manifestations, wait a couple of hours and give another 10 minims, and a third and a fourth at the same intervals. If no further symptoms appear, the large dose may be given.

If, however, the hemorrhage is severe, as shown by the large quantity of blood passed by the bowel, and even more if general symptoms of hemorrhage prevail, with or without the passage of blood—that is, sudden fall of temperature, pallor, cold sweat, cold extremities, rapid thready pulse, restlessness, and air-hunger, more decided measures must be taken. There is one drug that is of greater value than any other, namely, morphine.

It is to be given in doses of ¼ grain under the skin. Morphine is not a styptic, nor has it any effect on the caliber of the vessels or coagulation time, but what it does do is to afford the maximum amount of rest, both to the bleeding bowel and to the anxious brain, and to the restless and uneasy body, whose useless movements keep up the blood pressure. This gives the bleeding vessel a chance to do what it is trying to do—form a clot. It is pointed out that perforation occurs in these hemorrhages—one-fifth of the perforations; and that the use of opium blinds the symptoms, and no doubt it does, but, while anticipating such a complication, we cannot sit by and see our patient die from the hemorrhage.

There are many cases that come between these two extremes, and success in these cases is the fruit of experience and judgment. One can only say, when a patient is to be under constant observation for some hours to come, as he ought, hold off the opium as long as possible. If circumstances will not permit remaining by the patient, one should not spare the opium.

The bowels should not be moved for some three days after a hemorrhage, and then with caution. One should get back on the diet slowly and combat meteorism.

When the hemorrhage causes exsanguination, it must be treated like any other hemorrhage—elevation of the foot of the bed, heat to the extremities, saline infusions of 0.9 per cent. sodium chloride solutions, or transfusions.

Even more dangerous than hemorrhage is perforation. It occurs in 2 to 3 per cent. of the cases and causes some 12 per cent. of deaths in typhoid fever. It seems to occur as frequently in cases treated with the bath, but because of the lower mortality of the disease so treated, the percentage of death from perforation rises. If one in eight or more of the deaths in typhoid fever are due to perforation, it becomes a moral obligation to know the signs well. One will recall the sudden onset, with acute abdominal pain, like a bolt out of the blue; its paroxysmal character, the rapid rise in the leukocytosis, the two most important early symptoms, I think; then the

change in respiration, rapid pulse, distressed expression, local muscle spasm, rigidity, tympanites, with, perhaps, obliteration of the liver dulness.

Every attack of abdominal pain should mean perforation until continuous observation convinces otherwise, for thus its occurrence will not be overlooked. When it occurs, the surgeon should be summoned, an operation performed, and the perforation closed. If satisfied that pain has no such significance, then the ice-bag or stupes, or a light application of the cautery or small doses of bromides or codeine may be used; or, if more severe, small doses of morphine.

The cerebral disturbances are characteristic of this disease. In the early stages there may be much headache. This is best treated by the ice-cap. Sleeplessness is another common occurrence, and, as has been explained in considering rest, a symptom to be combated. Hydrotherapy properly applied mitigates this to a great degree. Small doses of bromides may be used, or trional in 15 to 20 grain doses, or chloralamid in 20 to 30 grain doses; but if it is marked and prolonged, morphine is the best drug to use. Delirium and stupor are best met by the baths. In all decided cerebral symptoms, help may be obtained from the tepid bath, with cold water poured over the head; from the ice-bag, from morphine, and, finally, when the delirium is great or the stupor deep, from lumbar puncture, an easy and innocent procedure. Draw off 20 c.c. at least, and more will do no harm, though headache may be aggravated by draining the cord. No delirious patient should be left alone for a moment. Restraint by sheets or other devices are sometimes needed.

Circulation. When the characteristically slow pulse of typhoid fever becomes rapid, going above 100 to 110 to 120, our concern is elicited. If the heart is weakening, we shall get a poorer quality of first sound, perhaps evidences of dilatation and mitral insufficiency, poorer quality of pulse, and a fall in blood pressure. A sufficiency of diet and the hydrotherapy do much to prevent this disaster. If, however, the condition supervenes, stimulants must be given: digitalis in the form of infusion (2 drams four times a day), watching carefully for evidences of its physiological action, which, when obtained, indicate its cessation for a period, as the drug is slowly eliminated. If it cannot be taken by the mouth, on account of gastric disturbance, or when the demand is more urgent, diaglen may be used hypodermically in doses of 10 minims every four hours, or strophanthin ($\frac{1}{60}$ grain) intravenously or intramuscularly; strychnine, too, may be tried, in doses of $\frac{1}{30}$ or $\frac{1}{40}$ grain, every four hours, by the mouth or under the skin, according to circumstances. Caffeine, sodium salicylate (3 grains) every four hours, given in the same way, is even more reliable.

In threatened circulatory collapse, the more rapidly acting stimulants, as camphor (3 to 5 grains) in 20 per cent. solution, in sterilized oil, or adrenalin (15 to 20 minims) may be used intravenously.

Alcohol requires a special discussion. Briefly I do not consider it a stimulant in the true sense of the word. Viewed from a chemical, pharmacological, or therapeutic standpoint, I think the weight of evidence is against its stimulating properties. It is hard to conceive that this member of the marsh-gas series, all of whose other members—alkanes, alcohols, aldehydes, ketones—are depressant, should furnish this one exception, ethyl alcohol; or two, ethyl alcohol and ethyl ether, which are stimulating. The results of animal experimentation are decided for the depressant effect of alcohol, while I am convinced that the trend of therapeutic opinion is increasingly in the same direction.

That the stimulating effect of alcohol on the mucous membrane of the stomach may reflexly stimulate the circulation is left sub judice; that alcohol has a food value is scarcely denied, and no doubt has contributed to the patient's strength when the food is insufficient; but neither of these effects are justification for the use of alcohol as it has been administered in typhoid fever. In the so-called typhoid state, characterized by cerebral excitation, sleeplessness, rapid pulse, dry skin, coated tongue, and subsultus tendinum, the picture, to my mind, is one of hyperexcitation of the nervous centres by the toxins of the disease. I believe that the improvement seen in this condition by the judicious use of alcohol is due to the sedative effect of the drug on these centres and not to stimulation. This conception confines the use of alcohol to this stage or state of the infection and decries its use where any of the functions are failing from exhaustion. Personally, I would rather not use it at all in typhoid fever, except in the condition just described, and then tentatively: not more than a half ounce of whisky or brandy every four hours.

The urinary tract is not to be neglected. In the early days of the disease especially, the patient's bladder may be overdistended. Close observation should be directed to this organ, and with the first evidences of retention, efforts should be made, by hot applications over the hypogastrium or by hot enemas, to provoke urination. If such measures fail, catheterization must be done, but should be done by the physician, to avoid by his care the infection that so frequently follows this procedure. Overdistention means stagnation, weakening of motility, and lowered resistance to infection by pathogenic organisms and invites a bacilluria. Bacilluria is a very real menace, more to the community than to the patient himself, as true cystitis or pyelitis of typhoid origin is rare. It occurs in nearly 25 per cent. of the cases, often in such numbers as to render the urine turbid. It is most common as the temperature begins to approach the normal and runs into convalescence. It probably arises by infection through the kidney, the bacilli finding a good culture medium in the urine, especially when the urine is of a low acidity and there is any stagnation, such as might result from neglect of overdistention.

The bacilli tend to disappear spontaneously, probably at such a

time as the urine becomes a less favorable medium and the improved tone of the bladder facilitates mechanical removal. However, the condition may remain for weeks, months and, in a few cases, for years. It has been shown that a high degree of acidity, and especially an increasing content of organic acid, inhibit their growth. The effort should be made to eliminate them, as a prophylactic measure, for absence of local symptoms make the convalescent an innocent menace to the community. Urotropin has been shown to have an astonishing effect on their growth, sometimes clearing up a turbid urine in a day or two. It may be given in doses of 5 to 15 grains for two or three days, a week, or continuously, beginning late in the disease and continuing into convalescence.

A good rule would be, beginning when the temperature approaches normal and continuing for a month after, to give 5 to 10 grains of urotropin three times a day for three consecutive days each week. When this does not clear up the bacilli, or a true cystitis prevails, bladder irrigations of silver nitrate, 1 to 5000 daily, or a saturated solution of boric acid, should be instituted.[1]

Perhaps the most promising field of specific therapy at this time is the vaccine treatment, the hypodermic use of the dead bodies of the typhoid bacilli, derived from the patient's own blood (autogenous) or from others (heterogenous). The results are not yet sufficiently definite or the details of procedure accurately enough determined to find a place in an article of this kind.

Drugs aimed at the disease itself, intestinal antiseptics, etc., have been left out of consideration, because it is believed that, on the one hand, no good results are to be obtained, while, on the other, harm may be done by the drugs themselves and by the neglect of the more useful measures advised, entailed by reliance on the drugs.

CONVALESCENCE. Convalencence is one of the most trying periods of the disease to deal with, both from the standpoint of the patient and the physician. On the part of the one is the impatience to relieve the gnawing appetite and be released from a confinement which has already dragged itself out to such lengths. On the part of the other is the haunting dread lest the intestinal lesions be not yet healed and that too early activity and too early feeding will induce perforation and establish a relapse.

Less difficulty will be met with in the matter of the dietary if sufficient food has been administered according to my advice, for hunger will be less imperative, and a considerable variety has already been allowed. During convalescence it has been demonstrated that a nitrogen storage takes place which does not obtain in health. We may then add slightly to our protein intake by allowing scraped beef, loin chops, and chicken. The kinds of food that we do avoid, however, are those with a large indigestible residue.

[1] Connell, AMER. JOUR. MED. SCI., May, 1909.

We can feed pretty much as follows, if our patient has been confined largely to a milk diet or has been highly toxic: After the temperature has been normal for four or five days, allow milk toast, then poached egg, then a chop or piece of chicken, then spinach, asparagus tops, purees of peas or tender string beans, the articles of the preceding days being also allowed. Watch the effect of each article carefully.

During the convalescence, excitement must be avoided, business cares excluded, too frequent visits from well-meaning friends interdicted, and other sources of fatigue prevented. After the patient has been restored to a considerable degree of strength, he should still be forbidden to return to work too early. A long vacation, if possible, of three to six months, should be taken, and it is often of great benefit if taken in other surroundings. After the temperature has been normal for a week or ten days, the patient may be propped up in bed, and in three or four days more be allowed to sit up in the chair, a little longer each day, and if all goes well, in a week be allowed to try his feet.

Every case must be approached on its merits, and if the pulse shows by its rate and poor quality that the demands on it are too great, the rest must be prolonged and the getting up be more gradual. To get the patient out in the sun and air is certain to facilitate the progress of his convalescence. During this time his bowels must be attended to with the same care as during his illness in bed, at first aiding with enemas, and later with a mild cathartic like cascara. If there is a great deal of anemia, Blaud's pills may be given, 5 grains, three times daily. If the weakness is prolonged, strychnine, $\frac{1}{40}$ grain, three times daily, may be administered.

A slight rise in temperature, day after day, is one of the most nagging features of a convalescence. This may be due to absorption from a neglected bowel, or it may be due to insufficient food, and eminent clinicians long ago pointed out that these patients' temperatures returned to normal when solid food was given them. Others still, who have been highly toxic, whose urea-forming function has been impaired, may become febrile if meat is given too early —the so-called "febris carnis." Others mend rapidly when kept in the air and light.

PROPHYLAXIS. Typhoid fever is a preventable disease. Its prevention awaits upon the overcoming of an inertia. McCrae divides the subject under three heads: (1) General measures, which are for the protection of the community; (2) special measures in connection with the patient; and (3) preventive inoculation. The knowledge that the disease is conveyed through the water, milk, food, especially oysters and green vegetables eaten raw, demand the supervision of the State and the community, through Boards of Health and Sanitary Commissions, supervision of the water supply, with reference to policing the water sheds, filtration plants, and all the

details dictated by sanitary science. It demands supervision of the milk supply, inspection of the dairies, health of the workers, and a consideration of sewage disposal with reference to oyster beds. It demands notification by physicians to Boards of Health of all cases of typhoid or suspected typhoid fever occurring in a community, and it demands education of the public through schools, lectures, and exhibitions of the part these various factors play in the prevention or spread of the disease.

In connection with the patient arises the question of isolation. Here in New York, as pretty much everywhere else in this country, typhoid fever is treated in the same wards as the general run of cases, and we assume from our familiarity with this condition of affairs that there is no danger in the practice. It is a little startling, then, to be told that the statistics of one of the best conducted hospitals in this country show that from 1.5 per cent. to 2 per cent. of the cases of typhoid fever treated in this hospital are of hospital origin. I have seen this thing occur in my own wards, and so has every attending physician, but still our patients are not isolated. As regards the disinfection of stools, urine, sputum, vomitus, and care of clothing and utensils, all this has been touched upon. If we were faithful in carrying out these measures in every case of typhoid fever, there soon would be no more cases, for, after all, the patient is the true source of typhoid fever.

The attendants about a patient are a source of infections for others unless conscientious in their cleanliness. A great source of danger that might be overlooked are the flies, carrying the infectious material direct on their legs and bodies from the excreta to food and water. Screens are an obvious help in their exclusion.

One great menace to the community, the handling of which is no easy problem, is the chronic bacillus carrier. Some patients harbor for months and years virulent bacilli in the urine, and more commonly in the feces, from constant passage into the bowel of bacilli from an infected gall-bladder. Bacilluria and its treatment have been touched upon. Some day, perhaps, Boards of Health will demand to know whether the stools of a typhoid fever patient are free from bacilli before he is allowed the freedom of the community, as they now demand to know whether the Klebs-Lœffler organism is absent from the secretions of diphtheria patients before release from quarantine.

As has been said, serotherapy has not yielded tangible results as yet, but much that is promising has come from the preventive inoculation or vaccination originated and applied to the troops of the British army by Sir A. E. Wright.

The procedure consists in the culture in bouillon of the organism for four weeks, then killing at 60° F. Different strains are mixed together, standardized, and two injections made at intervals of two weeks of amounts of 0.5 to 1.5 c.c., the first containing about

1,000,000,000 bacilli, the second 2,000,000,000. This is usually injected into the flank, from which may result some redness and pain and involvement of lymph glands draining that territory. There may be some constitutional reaction, fever, malaise, nausea and vomiting, but of no significance. Statistics seem to show that among some 20,000 inoculated soldiers the incidence of disease and the mortality was about one-half of that of the 150,000 uninoculated. This looks to be a tremendous advance in the struggle against the disease.

CHOLERA FROM A MODERN STANDPOINT.

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The recent outbreak of cholera in Europe has furnished reasonable and scientific evidence that the theories we have heretofore entertained regarding the period of incubation of this disease and the character in which it appears have been more or less erroneous. Reliable proof has been presented to show that, while it is probable that typical cases of cholera present themselves within five days after infection, there are mild or irregular cases, the incubation of which may cover a period of weeks, or, as in the case of cholera "carriers," symptoms of the disease may never occur, or when they do, it is usually after the administration of some form of intestinal stimulant sufficiently powerful to arouse the dormant organisms to activity, which is then followed by a sudden appearance of the symptoms of the disease. Even when this does not occur, it is reasonable to believe that carriers who remain apparently well may act as a medium of infection. If I remember correctly, a case of a cholera carrier has already been referred to by Professor Calmette. In this instance a young man, presumably infected in Hamburg during the outbreak of cholera in that city in 1892, traveled for two or three weeks on the Continent, and then, after a night's debauch, suddenly developed the disease.

A similar case has recently occurred in my service at the New York Quarantine Station, the facts of which are as follows: A vessel arrived at Quarantine from Naples after a voyage of fourteen days; the certificate of the ship's surgeon stated there had been no deaths on board, no cases of infectious disease or suspicious cases, nor any case of intestinal trouble during the voyage. None were found sick on the arrival of the vessel. As an extreme measure of precaution at the New York Quarantine Station, during the emerg-

ency above referred to, it was required that a list of all who had applied for treatment of any kind during the transit of the vessel be furnished by the ship's surgeon of all vessels arriving from cholera-infected ports or ports acting as the outlet of infected sections without regard to the character of the ailment. In this instance it was found that six men had applied for treatment for various causes while at sea; none, however, were treated either for diarrhœa or gastro-intestinal trouble of any kind. This group, all of whom were steerage passengers, were not under treatment on arrival, and were practically well. They were transferred to Hoffman Island for further observation. After two days' detention at the latter place, two or three of them were given 2 grains of calomel at bedtime, followed the next day by a cathartic. The following morning one of this group was in a condition of collapse, and died within twenty-four hours afterward. The character of the discharge from the intestinal tract became typical of cholera preceding the death of the patient. The autopsy did not reveal the cause of death. However, the bacteriological examination of the intestinal contents furnished prompt evidence of cholera. Specimens were not only subjected to exhaustive bacteriological investigation by Dr. E. C. Baldwin, bacteriologist of the New York Quarantine Laboratory, but also by Dr. William H. Park, bacteriologist of the Department of Health of the city of New York; and Dr. Anderson, Director of the Hygienic Laboratory of the U. S. Public Health and Marine Service at Washington, D. C., all of whom declared the case to be cholera.

Indisputable evidence has also been furnished that mild and irregular cases of cholera are not uncommon. A case of this character was recently found on a vessel arriving at New York from Italy. The patient was not very ill, and from the history the period of incubation could not have been less than sixteen days.

The fact that a cholera carrier and a mild case of this disease with a prolonged period of incubation occurred on two of the four vessels arriving at the port of New York, would indicate the frequency of these types of cases, and that the popular belief that symptoms of cholera appear within five days after infection is one which cannot be fully accepted.

In view of our present knowledge of cholera, the means by which it is to be dealt with in the future becomes a matter of great importance, particularly to those who are responsible for the public health. In the consideration of this subject it must be fully appreciated that modern quarantine, or the coast defence against the invasion of infectious disease, cannot offer full protection against the introduction of cholera into seaports or the interior. An attempt to carry out such a measure, while it would not secure the desired results, would go far toward destroying commerce, would be impracticable, and not in accord with modern sanitation, chiefly for the

reason that the period of incubation is uncertain, and the existence of cholera carriers has been practically proved. Reasonable and practical quarantine regulations will secure a thorough inspection of passengers and crews on incoming vessels and the detection of infectious diseases if they exist on board either in a mild or typical form, and will also include other valuable means of protection. However, the detention at Quarantine of those who have been exposed to cholera on shipboard, which has hitherto been looked upon as the chief means of preventing the introduction of this disease on this shore, must now be considered only as partial protection, because the assumption that any period represents the maximum time that cholera infection will develop in a person is entirely theoretical, and in view of the fact that long periods of incubation and also cholera carriers, exist. It would seem that the detention of suspects for more than five days, during which time typical cases would probably present themselves, is unreasonable and unjustifiable as a practical method of protection. This brings us to a realization of the fact that hereafter each community must be prepared to accept the responsibility which belongs to it in the protection of its people against this as well as other infectious diseases, and very forcibly suggests what practical sanitarians have long hoped for, that is, the presence in each town and city of a health officer or health officials who are practically and not theoretically familiar with infectious diseases, and who understand the importance of their early detection, either in the typical, mild, or irregular form—an exceedingly important factor in the control of outbreaks. Already, New York, Massachusetts, Pennsylvania, and other States have been working toward the consummation of this important object which in the future must constitute the logical and practical means of preventing the extension of infectious diseases.

Apparently what we have recently learned in regard to cholera would show that our defence against it is much weaker than we have heretofore believed. This is not the fact. There is an extremely optimistic side to this subject and one which has practical and scientific support. The belief has long existed that the ravages that cholera and other infectious diseases have caused in the East is indicative of what would occur if these diseases were to enter any community. This has been the keynote of statements frequently made by alarmists. This belief is without any reasonable or logical foundation whatever, and it may be safely stated that where modern sanitary regulations are in force such conditions will never occur. On the contrary, in the absence of a general infection, cholera does not extend rapidly, and where proper sanitary regulations are in force, should soon disappear. A general infection either on shipboard or on land is a serious and justifiable reflection

on whoever may be in charge of the health of those either on the vessel or in the community where the infection occurs.

During the outbreak of cholera which occurred in Naples in 1882, 800 to 1000 deaths daily was not an unusual occurrence. During the recent outbreak in the same place there has never been more than 20 to 25 deaths during the twenty-four hours, and the outbreak referred to continued only a month before it was officially declared at an end. There are two factors which are accountable for this: First, Naples now has a proper water supply, and no general infection has occurred; and, second, modern sanitary regulations are now enforced in a way which reflect great credit on those in charge of the Municipal Department of Health of that city. Cases of cholera have recently occurred in other European cities—Vienna, etc.—with little or no extension. No more effective or impressive evidence that cholera can be brought promptly under control, and does not extend where even ordinary care is used, is furnished in the case of two vessels which arrived at the New York Quarantine Station during October. On both a case of cholera occurred among the steerage passengers two days after leaving Naples, and death ensued within forty-eight hours. On each of these vessels there were over 1000 steerage passengers, which necessarily involved close and prolonged contact. The cases were suspected by the ship's surgeon, and were promptly removed from the other passengers, and, on their death, were buried at sea. Both vessels were afterward ten days in transit, and on arrival at this station the passengers above referred to were detained for an additional five days, but in neither instance did a secondary case occur. In both cases the disease was contracted before embarkation, and there was an entire absence of any ship infection. If this result can be secured on shipboard, it can also be obtained in a community where modern sanitary methods are in operation. Such evidence must receive the most careful consideration. As a matter of fact, ship infection is now practically a thing of the past. In former times, when sailing vessels, and probably small steamers kept their water supply on the deck, or some place accessible to everyone, there was but little reason why there should not be a general infection on shipboard. However, in modern passenger vessels it would be practically impossible for general infection to occur either in connection with the water supply or food. Neither will general infection follow cholera outbreaks on land if a proper water supply exists, and no community should enjoy the reputation of being under proper sanitary regulations which does not have this. It may be said that the same means of protection which are now employed to prevent general typhoid fever infection would be equally formidable against a general cholera outbreak.

As cholera carriers, mild and irregular cases of this disease may enter a community practically unannounced, it is necessary

that Departments of Health should prepare to deal with such an emergency, particularly at times when there is known to be danger of the introduction of cholera. A rigid investigation should be made of the death certificates which give as the cause of death gastro-enteritis or intestinal trouble of any kind. This should always include a bacteriological examination. Practising physicians should aid in carrying out this means of protection. The effect of this is to secure the early detection of cholera, either in a typical or irregular form, and when this is done, and in the absence of general infection, it is reasonably certain that no serious result will follow, so far as the extension of the disease is concerned.

It would seem reasonable that in the future we must recognize that isolated cases of cholera may appear anywhere which has known or unknown intercourse with the cholera-infected places, and the possibility of this must be recognized; but it must also be understood that while this is so, if general infection is guarded against and proper precautions are taken in individual cases, even the same that are properly carried out in the presence of typhoid fever, we may dismiss the idea of serious results to a community so far as the extension of the disease is concerned.

Bacteriological examination should always be included in determining the presence of cholera. This refers to the examination of alvine discharges or the material taken from the rectum by swabs. At the New York Quarantine Laboratory it has been found that nearly half of the general examinations of this kind, even where there was no clinical evidence or special reason to suspect cholera, showed curved bacilli of various sizes and shapes. In many instances these organisms resembled the cholera vibrios. Where the latter forms are numerous in this examination, the case should be regarded as suspicious until the cultural characters prove it to be otherwise. The presence of a few actively motile organisms in the hanging drop adds considerably to the suspicion.

In cholera carriers and mild cases of this disease the cultures may show the vibrios even though the smear shows very few suspicious organisms. It is usually only in the active cases that the smear and hanging drop, made directly from the discharges or rectal swab, will afford such unmistakable evidence as is required to pronounce the case true cholera from the point of view of the bacteriologist. In all other cases it is necessary to isolate pure cultures and prove them cholera vibrios or otherwise by suitable bacteriological tests.

THE RELATION OF DISEASE OF THE GALL-BLADDER AND BILIARY DUCTS TO THE GASTRIC FUNCTIONS.

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The effect of certain pathological conditions of the stomach upon the physiological functions of the stomach has engaged the attention of physicians for a number of years. Gastric ulcer, with the associated hyperchlorhydria, is well known. It is only within the past half-decade, or more, that we have begun to realize that a lesion anywhere in the gastro-intestinal tract, or its associated organs, may disturb the gastric functions. We now hear of "acid appendix dyspepsia," of "flatulent gallstone dyspepsia," etc. This has, no doubt, resulted from the very active surgical interference in diseases of the organs of the abdomen, especially of the upper abdomen. The surgeons, among whom are principally Moynihan, Mayo Robson, Paterson, the Mayos, Maurice Richardson, and Deaver, were the first to write of these dyspepsias. My attention was called to this relation some years ago, but more definitely since the meeting of the American Gastro-enterological Association in Boston, where our present presiding officer read a very suggestive paper upon the association of cholelithiasis and hyperchlorhydria.

In looking through the histories of my own cases which bear upon this subject, I have found 249 patients in whom the diagnosis of some disease of the gall-bladder and ducts had been made; and in 258 patients in whom the diagnosis of appendicitis had been made a large percentage had a positive disturbance of the secretory as well as of the motor function of the stomach. In this paper I wish to refer more particularly to the relation between gall-bladder and duct disease to disturbance of the gastric functions, and I shall, therefore, analyze rather closely the 249 cases of gall-bladder and duct affections. In 156 of these cases gastric analyses were made with the usual methods employed clinically to determine the secretory and motor condition of the stomach; 84 (54 per cent.), had a hyperacidity, 31 (20 per cent.) had a subacidity, and 41 (26 per cent.), had a normal secretion. The motor function, so far as could be determined, was normal in about the same per-cent. of cases. Of the 249 cases upon which these observations were made, 51 cases came to operation; 30 of these had gastric analyses made; 16 (about 53 per cent.) had a hyperacidity, 8 (about 26 per cent.) had a subacidity, and 6 (about 20 per cent.) had a normal acidity. In a number of the 249 cases the gastric analyses

were made because there were definite symptoms of gastric disturbance associated with the gall-bladder symptoms, but in some it was made because the gastric symptoms were the only ones present, and later, when treatment did not produce the desired results, the abdomen was opened and either gallstones were found, or adhesions from a local peritonitis, due to gall-bladder infection, had occurred between the gall-bladder and duodenum, between the gall-bladder and stomach, or both. In several cases both gallstones and gastric ulcer were found.

	No.	Hyperacid.	Normal.	Hypoacid.
Total gall-bladder cases . . .	249			
Cases with gastric analyses . .	156	84 (54%)	41 (26%)	31 (20%)
Total cases operated on . . .	51			
Cases with gastric analyses . .	30	16 (53%)	6 (20%)	8 (26%)

The number of cases upon which these figures are based is, of course, not large, but I have had access to the statistics of other men, and my experience does not seem unusual. My brother, Dr. M. J. Lichty, of Cleveland, gives the following figures. Of 36 gall-bladder and duct cases, 28 had gastric analyses; 19 had a hyperacidity, 5 had a subacidity, and in 4 the secretion was normal.

From these figures it would seem that about 75 per cent. of all gall-bladder cases may be associated with a disturbance of gastric secretion, and of this 75 per cent., two-thirds have hyperchlorhydria. Gastric motility, as well as gastric secretion, was disturbed in about the same proportion of cases. In these days much is said about disturbance of gastric motility and of its relative value clinically compared with gastric secretory disturbances. It is scarcely necessary to draw such distinctions, because the two may frequently be associated in some cases, and, in others one may predominate, while in still others the other predominates. When the statement is made that gastric analyses are misleading, that the motility of the stomach is more important than is the secretory function, it gives evidence that the observer has not made many complete gastric analyses, or that he has misinterpreted his findings.

In presenting these cases, I must necessarily omit a narration of the details of dyspeptic symptoms which pointed to gastric secretory and motor disturbances, but which are not usually mentioned in text-book descriptions of gall-bladder and duct affections. My main object is to show that certain gastric disturbances are rather constant in these cases, and, if persistent, they may lead one to a diagnosis of the real condition, independently of such pronounced symptoms as jaundice and biliary colic.

Rolleston[1] says: "Indigestion is a frequent result of gallstones;

[1] Disease of the Liver, Gall-bladder, and Bile Ducts.

so-called irregular biliary colic not infrequently manifests itself as dyspepsia, and may be due to adhesions between the gall-bladder and stomach" (or duodenum). But later he says that during the attacks of biliary colic the amount of HCl is either normal or diminished. I think myself, in the height of the attack of biliary colic the secretions may be subnormal, but between the attacks they are more often increased. In some of my cases, repeated examination of the vomitus, during an attack of biliary colic, showed an absence of free HCl and a low total acidity, but the gastric analysis during the interval always showed a hyperchlorhydria. The following case illustrates this well:

Mrs. S., aged twenty-nine years, has had nausea and vomiting for the past five months, but no pain. Four months ago she had a similar attack. A year ago, with such an attack, she had fever, which lasted two weeks and was thought to be typhoid. In the interval the patient had burning and gnawing in the stomach two hours after meals, which was relieved by taking food. Eight years ago the patient had jaundice. At the age of nine years she had a severe attack of typhoid fever. The analysis of the vomitus during the attack for which she consulted me showed no free HCl and a total acidity of only 12. After the attack had subsided, and when she was on her usual diet, an Ewald test breakfast was given and the free HCl was 24, combined 40, and total acid 68. At the operation the gall-bladder was found thick, white, and large, with a stone in the cystic duct. There were no adhesions. The appendix, which was thick, large, and bound down with adhesions, was removed.

It is a matter of observation by many that a patient who has gallstones may give a history of being disturbed in his sleep about 3 A.M., with discomfort or pain in the epigastrium, similar to the discomfort which a patient having a hyperchlorhydria or a so-called nervous dyspepsia experiences.

Moynihan[2] in an article upon appendix dyspepsia, refers to the occurrence of hyperchlorhydria, or acid dyspepsia, in association with many cases of chronic appendicitis. My experience in over 258 cases of appendicitis, of which, 127 were operated upon and 111 had gastric analyses made, confirms Mr. Moynihan's experience. In the last few years I have called the attention of some of my medical and surgical colleagues to these facts, and all seemed to be of the opinion that there are nearly always rather definite symptoms of gastric disturbances associated with disease of the gall-bladder and ducts, and also with disease of the appendix.

There seems, therefore, a rather uniform opinion that gall-bladder and duct disturbance is associated with symptoms of dyspepsia, such as are usually referred to some disorder of gastric

[2] Brit. Med. Jour., January 29, 1910.

function. The question now arises, Is there usually a certain demonstrable alteration of gastric function, that is, of secretion and motility, in these cases, and if there is, is it secondary to pathological lesions elsewhere in the gastro-intestinal tract, or is it merely a coincidence?

From the experiments of Pawlow, Starling, Cannon, and Murphy, we can very readily see how a lesion in the gall-bladder and ducts, especially when it has caused local peritoneal adhesions, might produce a pylorospasm and thus disturb both the secretory and motor function of the stomach. It is now generally accepted, especially since Cannon's experiments, that the dominant control of the pylorus lies on the duodenal side of the pylorus, and any disturbance there, such as of the biliary, pancreatic, or duodenal function, may disturb the normally well-balanced mechanism of the pylorus. Cannon and Murphy[3] have shown in cats, by an end-to-end anatomosis high in the intestines, that food will leave the stomach only after five or six hours, whereas normally it leaves within ten minutes. Moynihan, in the article on appendix dyspepsia already referred to, suggests that the action of the pylorus as shown by these experiments seems to be protective, and in this way explains the effect of a lesion of the appendix upon gastric function.

I have attempted in the last year or more to determine whether any change in gastric function would occur in dogs in which an artificial lesion of the gall-bladder, corresponding to cholelithiasis in the human subject, had been produced. The primary object of these experiments was not so much to determine specifically the relation of gall-bladder disease to gastric function as to attempt to find a pathological basis, or explanation, for some of the great number of cases of so-called hyperchlorhydria which are now being recognized by both the internist and the surgeon. It seems to have been the tendency, until recently, to look upon patients who have a gastric hypersecretion, but no definite symptoms of gastric or duodenal ulcer, as being simply neurotic and worthy of only scant consideration. I find myself guilty of this error as I review the histories of some cases which had a gastric hypersecretion, for not infrequently have I found these patients (who gave definite symptoms of gastric hypersecretion, and in whom the condition was verified by gastric analyses) suddenly develop definite gallstone or appendix symptoms, and at the operation reveal pathological conditions which must have been the foundation for the whole train of gastric symptoms.

The experiments upon dogs referred to were conducted in the laboratories of the Columbia Hospital with the assistance of Dr. John W. Dixon, assistant surgeon to the hospital, and Dr. Florence Kline, in charge of my private laboratory, to both of whom I wish

[3] Annals of Surgery, 1906, p. 512.

to acknowledge my indebtedness for surgical and laboratory technique which I had neither the ability nor time to perform myself. We took six healthy dogs of average size and determined as nearly as possible their normal gastric function after a test meal similar to the Ewald test breakfast usually employed clinically. Gastric secretion, as well as motility, was observed as nearly as possible as it is in man. The technique was as follows: Two ounces of bread was thoroughly dried and broken into small pieces and soaked in eight ounces of water, so as to make an emulsion which could be easily injected through a stomach tube. A dog, after having fasted for ten or twelve hours, was given this portion through a stomach tube which had been passed into the stomach. At the end of thirty or forty minutes the stomach was aspirated by inserting the stomach tube with a politzer bag attached. The contents thus recovered was filtered and the acidity estimated by titrating with a decinormal sodium hydroxide solution.

A number of tests were made in this way until the results became rather constant, or until a fairly definite average was obtained. At a sufficient time after the dog became accustomed to his new surroundings, and also to the passing of the tube to determine his individual acidity and motility, the abdomen was opened under anesthesia and a foreign body, usually a hard cinder from the furnace room, which had previously been dipped into a culture containing streptococci, or mixed microörganisms, was inserted into the gall-bladder, which was opened and again carefully closed and returned into the abdominal cavity. After the dog had recovered from the immediate effects of the operation and was again upon a general diet, the analyses of the stomach contents were resumed, and, after a certain time, a comparison made between the gastric analyses before and after the operation. Five dogs were operated upon and foreign bodies inserted into the gall-bladder, the sixth being kept as a control.

Dog No. 2, after having twenty-three test breakfasts given in the course of thirty-one days, showed an average acidity represented by 23.7, and was operated upon, under ether anesthesia, January 19, 1910. The gall-bladder was delivered through a Kocher's incision, opened, and a rough cinder the size of a pecan was inserted. The gall-bladder was closed and returned to the abdominal cavity. Recovery was satisfactory. On January 25, when the dog was taking his usual diet, the test breakfasts were resumed. It was very soon found that the stomach did not empty as promptly as before the operation. In sixty-five days twenty-seven test breakfasts were given, showing an average total acidity of 28, or a gain of 4.6 points. On March 25, 1910, the abdominal cavity was again opened for the purpose of producing a lesion at the cecal pouch (appendix). The gall-bladder was found very much shrunken and tucked up as far beneath the liver as possible.

Dog No. 4 had thirty-two test meals in forty-three days, with an average acidity of 21. Was operated upon, February 9, 1910. A cinder, which had previously been dipped into a culture of streptococci, was inserted into the gall-bladder with the usual technique. In the course of sixty days twenty test breakfasts were given, with an average acidity of 19. On April 1, 1910, the abdomen was opened, and a few firm adhesions were found binding the omentum to the gall-bladder.

Dog No. 5, after twenty test meals in twenty-eight days, showed an average acidity of 22.8. Was operated upon, February 10, 1910, A cinder infected from a mixed culture was inserted into the gall-bladder. Following the operation the wound in the abdominal wall was opened and there was a marked local infection, which, however, finally granulated over nicely. After the dog had entirely recovered from the operation, twenty-four test meals were given in sixty-three days, with an average acidity of 33.3. On April 15, 1910, an exploratory abdominal operation was done, and extensive adhesions were found between the gall-bladder, omentum, and abdominal wall.

Dog No. 6 had thirty-one test meals in forty-one days, with an average total acidity of 21.8. Was operated upon, March 18, 1910, and a cinder infected with pus from a patient having puerperal septicemia was inserted into the gall-bladder. The incision healed without any infection. After recovery from the operation twenty-five test meals were given in sixty-eight days, with an average total acidity of 24.5.

Dog No. 7 had fourteen test meals in fifteen days, with an average total acidity of 24.6. Operated on February 25, 1910, and a cinder infected from a streptococcus culture was inserted into the gall-bladder; the wound closed without any infection. After the operation twenty-one test meals were given in fifty-nine days, with an average total acidity of 37.5, or an increase of 13 points. On April 22 the abdomen was again opened for the purpose of examining the gall-bladder and producing a lesion in the appendiceal pouch. The gall-bladder was very small and high under the liver. It was bound down by a great many tense bands of adhesions.

The average results of the analyses of the gastric secretion are as follows:

	Before operation.			After gall-bladder operation.		
	Test meals.	Days.	Average acidity.	Test meals.	Days.	Average acidity.
Dog No. 2	23	31	23.7	27	65	28.0
Dog No. 4	32	43	21.0	20	60	19.0
Dog No. 5	20	28	22.8	24	63	33.3
Dog No. 6	31	41	21.8	25	68	24.5
Dog No. 7	14	15	24.6	21	59	37.6
Dog No. 8	24	58	16.7			

It will be seen that in Dogs Nos. 2, 5, and 7 considerable change in the gastric secretion took place. The motility also was disturbed. When these experiments were begun, it was intended to aspirate the stomach an hour after the test meals had been given, as is done clinically, but we soon learned that the gastric motility in the dog was more rapid than in man; we, therefore, withdrew the contents in thirty minutes, and even then sometimes did not obtain a satisfactory amount. After the gall-bladder operations this was suddenly changed, and at the end of thirty and even forty and fifty minutes abundant amounts could be aspirated. This change was so evident and constant that we concluded the motility of the dog's stomach was decidedly disturbed as the result of the pathological conditions which the foreign body in the gall-bladder had produced. The change, or delay, in motility seemed to be in direct proportion to the extent of the lesions produced.

It seemed to us with this technique we were able to produce conditions and determine results in the dog very nearly as they are found clinically, and that we had, as nearly as possible, met the necessary requirements for conducting an experiment. We, of course, appreciate that the Ewald test breakfast does not give us scientifically correct information of gastric function, and yet it is the method generally employed clinically. The Pawlow artificial second stomach pouch, or cul-de-sac, was not employed in these experiments, because we were of the opinion that a disturbance of the normal gastric secretion and motility would be caused by the operation itself and the consequent adhesions. We hope to confirm this opinion by experiments which are now being conducted in our laboratory.

Our technique might be objected to on the ground that a meal forcibly injected into a dog's stomach would not excite the amount of secretion or degree of motility occurring after a meal which the dog had taken with his proverbial relish and greed. But the object of the experiment was not so much to determine the normal gastric secretion in dogs as to determine whether the constant secretion and motility found by a certain technique would be disturbed by producing a lesion in the gall-bladder and ducts.

We attempted to check up our experiments by having a sixth dog as control in the same kennel with the dogs which had been operated upon, whose stomach contents were examined every few days, but who was not subjected to operation. We very soon learned that a number of factors, such as irregularity of feeding, disturbance of health and comfort, segregation of the dogs, and the introduction of a keeper who was not agreeable to them, influenced gastric secretion quite decidedly, but we attempted in every way possible to obviate these factors, and we feel that our results are as reliable, if not more so, than those obtained clinically.

I feel, therefore, inasmuch as my own clinical experience, together

with that of the large experience of other clinicians, seems to be confirmed by the experimental evidence which I have attempted to present, that I might safely draw the following conclusions:

1. A lesion of the gall-bladder and ducts may disturb the gastric functions.

2. This disturbance most frequently consists of a hypersecretion of gastric juice and a diminution of gastric motility, and may be in direct proportion to the lesions present.

3. So-called hyperchlorhydria, with its accompanying symptoms, should be looked upon as an evidence of some definite pathological lesion somewhere in the gastro-intestinal tract or its appendages, and should be treated symptomatically only when organic disease can be excluded with a satisfactory degree of certainty.

THE DYSPEPSIA OF OLD AGE.

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For a number of years certain digestive disorders in old people have come under my observation, and in these the cause seemed purely dependent on the changes incident to advanced years. The manifestations of senility shown by atheromas, degenerations, ossifications, calcifications, etc., have been discussed at length by contemporary medical writers, but some of the strictly gastro-intestinal distubrances have received scant notice, being regarded more as pathological features of maturity than normal accompaniments of old age. Gereology we find laid down already, and Dr. Nascher, of New York, recently suggested the term geriatrics to cover the same field in old age that pediatrics covers in childhood.

It may be of interest, therefore, to consider some of the dyspepsias of this advanced period of life as radically different from those occurring during the height of adult vigor, while normal metabolism should be waging an equal warfare between the constructive and destructive forces. According to Fenwick's investigations, out of every 100 cases of chronic dyspepsia in persons over sixty-five years of age, 66 are secondary to organic diseases of some important organs of the body, while 34 owe their symptoms to a progressive degeneration of the secretory structures of the stomach and intestines.

The stress of over sixty-five years of bodily activity will, in a majority of cases (at least two-thirds), result in disease of either the kidneys, prostate, heart, lungs, liver, pancreas, or a chronic gouty condition, which will set up a chronic asthenic gastritis; while in a

fair proportion of this number (possibly 10 per cent.) the dyspepsia dates back to long-continued hypersecretion due to gastric or duodenal ulcer, gallstones, or a diseased appendix. In the remaining one-third—those to whom this study principally applies—no marked organic impairment can be located, but the general assimilative powers decline, the body assimilates less organic matter, and destroys less inorganic matter, bringing about a waste of tissue and an increase of salts. Even should there be no general arteriosclerosis, abdominal arteriosclerosis may be present, giving rise to ill-defined digestive disorders, angina, and epigastric discomfort. Again, there are certain obscure dyspepsias, in which the arteriosclerosis is probably confined to the intestines, and in which such diagnoses can be made only by exclusion, the main indication, however, being that the gastro-intestinal disturbances are not dependent so much on the quality of the food as on the quantity.

Admitting that our information concerning these senile dyspepsias is in a somewhat chaotic state, it would seem permissible, and perhaps helpful, to divide them into two classes, which will be readily recognized clinically. These classes may be termed the *hyperkoric* and the *akoric*.

In the first, or hyperkoric cases, the degeneration and insufficiency of the digestive organs simply keep pace with the rest of the body. With decreased muscular activity there are decreased assimilative powers, and the patient finds that articles of food now disagree which were formerly digested with ease.

I have in mind at present a gentleman, now deceased, who informed me that up to sixty years of age he hardly knew he had a stomach. Possessing the appetite of a cormorant and the digestion of a rhinoceros, he was much surprised when suddenly, following neither illness nor change in habits, he realized that his appetite was diminishing, and that if he ate more than it dictated he would suffer. This hyperkoria completely revolutionized the last fifteen years of his life. He was apparently a hale old man, few of his daily habits except those of eating being changed, but he learned by several painful experiences that his digestive capacity had become curtailed, and for the rest of his life, which was terminated by an acute dysentery, he ate only about one-fourth of the amount formerly required. The only clinical symptoms I could ever elicit in this interesting case were intermittent tenderness over the abdominal aorta and occasional epigastric pulsation.

A large proportion of persons over fifty years of age show a diminution of free hydrochloric acid; a test meal revealing hydrochloric acid content as high as 25 becomes a decided exception, unless the oxyntic cells are subjected to some constant irritant. Pawlow tells us that the hydrochloric acid as poured out by the peptic glands is of a constant percentage value, by which he means that in each individual each gland secretes a juice possessing the same degree of

acidity, never varying. Admitting this, the gross findings would indicate a diminution in quantity of gastric juice, probably shared in by all the other intestinal juices. Thus we have different degrees of hypoacidity reaching to complete achylia gastrica. When this condition obtains the sense of hunger and thirst are wisely obtunded; the palate no longer beckons to gastronomic delights; the olfactories, once the psychic outposts of the digestive system, cease their agreeable suggestions; the digestive glands respond but tardily to the secretagogues, which formerly incited such joyous action; while the "appetite juices" well up but lazily from their tiny fountains. It is then that the old man realizes what Solomon meant, when, in describing old age, he wrote "desire shall fail," and the hungry tramp with his gnawing stomach becomes an object of real envy.

The hyperkoria, or early sense of satiety, is Nature's protection, and the dyspeptic manifestations simply indicate that the digestive machinery is performing its tasks haltingly and imperfectly. The machinery itself may be intact, but the dissolving and transforming juices are too scanty for efficient work.

The second class, or akoric dyspepsia, is generally found in obese old people, or in those in whom the mental edge, perhaps once sharp and bright, has been either blunted or worn off. There is more than habit in obesity, and the lessening activity is not so much the cause as the result of it. It is not lack of exercise that induces the corpulence of advancing years, nor is a morbid appetite an indication of renewed vigor. Both are evidences of declining vitality, and of the failure of that steady poise of maturity, that competent metabolic adjustment, so aptly termed by Herbert Spencer "the moving equilibrium." Assimilation no longer equals its calls; the departments of construction and repair are not now aggressive, and the task is to keep what has been won. The normal tonicity of the stomach gives place to relaxation, its walls become flabby, and more food is required to give the sensation of comfortable fulness. Pyloric insufficiency is generally present, while atonic constipation supervenes almost as a matter of course. Under such circumstances the lot of the aged sufferer is sad. Hunger cannot be satisfied, elimination is imperfect, and the intestines veer between obstinate constipation and lienteric diarrhœa.

There came recently under my observation an instance of this sort—a gentleman, highly educated, and at one time a prominent man of affairs. Having retired from business, he purposed spending his declining years in ease and quietude, and all went well until his family noted the development of an insatiable appetite, coupled with a slight dulling of his mental faculties. Although over seventy-five years of age, he showed no marked organic change, except a dilated stomach, and I was forced to the diagnosis of a senile akoric dyspepsia, with probable intestinal arteriosclerosis.

Apart from the lientery so frequent in this class of cases, there

is often the mitigating presence of a compensatory diarrhœa, which comes on at regular intervals, washing out the stagnated products of katabolism, and with each visitation affording a new lease on life.

The management of these two classes of senile dyspepsia naturally differs. In the hyperkoric dyspepsia we generally find that the food ingested furnishes barely enough calories for daily existence, allowing none for repair or "lost motion." So many articles have disagreed in the past that there is frequently a sitophobia, or fear of food, so that in many cases insufficient nourishment indirectly hastens the senile decay.

As physical activity increases combustion and waste, active exercise should be limited. The food should contain a minimum of non-digestible, non-nutritious elements and lime salts. Most of the predigested foods on the market are unavailable on account of their high percentage of alcohol, but the meat juices, peptonized milk, malted milk, lactone buttermilk, malt extracts, eggs, and the farinaceous foods may usually be given, to which, if deemed advisable, the gastric and pancreatic ferments may be added. The proportion of cellulose, which should properly enter into the dietary of adults so as to promote peristaltic activity, may be omitted from consideration, and all foods should be given in the most concentrated form, and in the shape most easily assimilated.

The principles of physiological economy, so carelessly followed during the period of anabolic excess, must now be rigidly observed; like the man whose earning capacity being diminished, has to adapt his methods of living to a depleted income, so constant care should be exercised, on the one hand, not to overtax the enfeebled digestive powers, and on the other hand, to avoid the pitfalls of caloric bankruptcy.

The management of the akoric type of senile dyspepsia will call for infinite tact and discretion. To curb the cravings of a second childhood, to regulate wisely an imperious will, untempered by judgment, to appease the gnawings of a constant bulimia, without overwhelming the intestines with undigested food, will demand a study of each individual. Should the teeth be bad, or mastication imperfect, soft or previously comminuted food will be advisable, and the aching gastric void may be at least partly filled with liberal potations of weak tea, or hot water flavored with sugar and milk. Between meals chewing gum will frequently assuage the pangs of hunger, and, unless there are esthetic objections, it may be indulged in ad libitum.

Alcohol has but little place in these conditions, either hyperkoric or akoric, and the remarkable instances of youthful old people, sustained and soothed by certain brands of whisky, and so graphically portrayed in the secular press, are generally the perfervid productions of paid advertisement writers. Cathartics should be used with caution, and gentle methods for cleansing the bowels should be combined with measures to overcome subsequent weakness.

The treatment of incidental complications need not be touched here. These complications are rarely avoidable, and often end the scene very quickly. In the department of the bodily upkeep, senile changes may be retarded, but their progress is sure, their termination fixed.

The only human agent on record who ever stopped the flight of time was Joshua, and even he did not turn back the clock. So, while we cannot administer from a spoon the elixir of youth, we can in many ways aid our timeworn patients in the task of growing old both gracefully and comfortably.

THE DIAGNOSIS OF TUBERCULOSIS OF THE BRONCHIAL GLANDS.

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In view of the fact that pulmonary tuberculosis is almost always secondary to tuberculosis of the bronchial glands, and as we know that months, even years, may elapse before the lung becomes involved, it is surprising that the diagnosis of bronchial gland tuberculosis has not received more attention. Except for a short paper, presented by Gray,[1] two years ago, which dealt chiefly with vertebral bronchophony, the subject has never been discussed by the National Association for the Study and Prevention of Tuberculosis; nor was it considered at the recent International Congress of Tuberculosis. Yet disease of the glands frequently gives symptoms, and often physical signs, months before the lung is invaded.

The lymph nodes, known as the tracheobronchial or mediastinal glands, are divided into three groups. The tracheal glands, which surround the trachea, are in relation with the superior vena cava, and the pneumogastric and recurrent laryngeal nerves. The group situated at the bifurcation of the trachea, are in contact with the pneumogastric nerve, and also with the pericardium anteriorly, and the œsophagus posteriorly. The pulmonary vessels are below, and the spinal column, adjacent vessels, and nerves on the right. The hilum glands extend along the bronchi to the lungs, and are in relation with the branches of the bronchial and pulmonary vessels.

The glands at the bifurcation of the trachea are usually the first to be diseased, Wollstein[2] finding the gland adjacent to the right bronchus the seat of the primary lesion in 74 per cent. of her cases.

[1] Proc. Nat. Assoc. for Study and Prevent. of Tuberculosis, 1908, p. 300.
[2] Arch. Int. Med., 1909, No. 4.

The individual glands rarely exceed an inch in diameter, and are usually smaller. As the trachea extends downward, it inclines to the right, so that at its bifurcation at the level of the fourth or fifth dorsal vertebra, it is near the right border of the vertebral column.

It is unnecessary in this paper to take up the pathology of tuberculous glands. It suffices to state that they may harbor tubercle bacilli when there is neither macroscopic nor microscopic evidence of their presence. The bacilli may remain for a time in a dormant, or so-called "lymphoid state."

The bronchial glands are much more prone to caseous degeneration than the cervical or mesenteric, in which the lymphoid state is often of long duration. When the process subsides, the glands frequently become calcareous.

Symptoms. The onset is usually insiduous, Schossman[3] stating that "the lack of cause for the change in the general condition of the child is truly characteristic of tuberculosis of the bronchial lymph nodes."

The symptoms are in part due to the absorption of the toxins of the tubercle bacilli, and also to the pressure of the glands upon vascular, bronchial, and nervous tissues. The toxic symptoms are familiar to us all. They are poor or capricious appetite, fatigue, especially in the morning, languor, irritability of temper, and irregular pyrexia. Dautwiz[4] in his exhaustive monograph on bronchial gland tuberculosis, mentions as an early symptom pain of indefinite origin, felt within the chest, which is brought on by deep breathing or vigorous physical exercise. Sometimes there is a substernal pain. Krämer[5] speaks of a constant pain in the mammary region, associated with interscapular dulness, both of which disappear under tuberculin treatment. Huskiness of the voice may be present. Schick[6] has called attention to the expiratory stridor of infants with enlarged bronchial glands. This resembles somewhat the expiration of asthma, but differs in that it is more often constant than paroxysmal. This sign, which is rarely present after the fourth year, is usually preceded by a few weeks of coughing, commonly diagnosticated bronchitis. When the child is resting quietly one observes that the stridor occupies the whole of expiration. Following excitement or a hard coughing spell the respirations may be normal for a time. In advanced cases the breathing becomes labored as the stenosis increases. These severe forms have been mistaken for laryngeal croup, and intubation has even been performed. This mistake can be avoided if one remembers that the voice is lost in diphtheria but not in bronchial gland enlargement. Under the term "status lymphaticus,"

[3] Diseases of Children, 1908, ii, 603.
Beitr. z. klin. d. Tuberkulos, 1909, xiv, 335.
[4] Beiheft z. med. Klin., 1908, iv, No. 9.
[5] Wien. klin. Woch., 1910, xxiii, 153

Friedländer[7] and Rachford[8] have reported cases whose clinical histories are quite typical of tracheobronchial adenopathy. In Friedländer's case the stridor was inspiratory. Most of the infants whom Schick observed with expiratory stridor died subsequently of tuberculosis.

Eventually a cough begins, and it is because of this symptom that medical advice is usually sought. The typical cough is paroxysmal, "brassy," and quite characteristic. While it often suggests the cough of pertussis, it is not always so severe. I have heard it when it suggested a cavity in the lung. It is of reflex origin, depending upon the pressure of the swollen glands on the vagus nerve. As there appears to be no explanation as to how this reflex takes place, the following hypothesis is suggested:

Cough is usually incited by the stimulation of the terminal filaments of the superior laryngeal nerve, a branch of the vagus, which ramifies in the mucous membrane of the larynx. The vagus is not a sensory nerve below its superior laryngeal branch. MacKenzie[9] has shown that a long-continued stimulation may set up a condition of irritability or hypersensitiveness in nerve centres. It is possible that such a condition of irritability is brought about in the ganglion of the vagus trunk by the pressure of the glands, and that as a result the superior laryngeal nerve, which arises from the inferior ganglion of the vagus, is stimulated, and a cough results. This hypothesis would explain the cough of pleurisy, and the tickling in the throat complained of by nearly all incipient cases of tuberculosis. It is possible that the cough of pertussis is occasioned in like manner.

Long-continued pressure may cause erosion of the trachea or bronchi, and the aspiration of the ruptured gland may be followed by a pulmonary lesion.[10] The œsophagus may in like manner be invaded, and fatal hemorrhages have resulted from the erosion of bloodvessels when the lungs have been free from disease.

Fisher[11] reports the case of a boy, aged five years, who died from this cause, and Barry[12] tells of the death of a woman, aged seventy-one years, whose caseous bronchial gland had caused a rupture of the vena cava. At the autopsy of a man, aged twenty-eight years, who died with symptoms of hemorrhage, Griffin[13] found in addition to a pleurisy with effusion, intense congestion of the trachea at its bifurcation, and an enlarged and caseous bronchial gland surrounded by a coagulum the size of an English walnut. The pretracheal glands were moderately enlarged, necrotic, and the seat of numerous hemorrhages.

[7] Archiv. Pediatrics, 1907, xxiv, 490.
[8] Amer. Jour. Med. Sci., 1910, cxl, 550.
[9] Symptoms and their Interpretation, 1909, Chapters iii, iv, v.
[10] Hall, Amer. Jour. Med. Sci., 1899, cxviii, 185, has reported a case in which a large piece of the bronchial gland was coughed up, with relief of symptoms.
[11] Lancet, 1909, i, 28.
[12] Med. Rec., 1891, xxxix, 166.
[13] Med. News, 1885, xlvii, 236.

Another patient of Fisher's,[14] a girl, aged seven years, who was in good health, and in whom there was nothing to suggest pulmonary tuberculosis, spat up blood from time to time for over three years.

It seemed desirable to ascertain the reliability of a number of signs said to be indicative of tuberculosis of the endothoracic glands. To this end 175 children have been studied; the results in each instance were compared with the skin tuberculin test—the undiluted "old" tuberculin being used. Some of the children had entered the Hartford Hospital for operation for adenoids and hypertrophied tonsils, and through the courtesy of Dr. Frederick R. Crossfield, I was permitted to examine them. I am indebted to Dr. Albert R. Keith for the opportunity of studying a number of the children at the Hartford Orphan Asylum. The remaining cases were scholars at our Preventorium, or Outdoor School.

Inspection. We observe in the first place, that the child with tuberculosis of the bronchial glands is usually frail, but this is not always the case, and Dautwiz very truly says that the condition "may hide back of the rosy cheeks of a happy, contented child, who is kept in good physical condition."

Seventy-seven out of 146 of our children in whom the appearance was recorded, were noted as being frail, and of these 57 per cent. reacted to tuberculin; 69 children who were not so classified, but included the robust and "indifferent," were tested, and 32 per cent. reacted; 59 children positive to tuberculin, who were weighed, were on the average twelve pounds under weight for their ages.

One should next look for the presence of dilated veins over the anterior aspect of the thorax. These veins were present in 92 out of 173 cases in this series, 50 per cent. of whom reacted to tuberculin. The presence of dilated capilliaries in the region of the seventh cervical vertebra seems to be a sign of less value. Slight puffiness of the face and eyelids from venous obstruction may be present.

One sometimes observes slight inequality in the expansion at the apices. This is best detected by looking down over the anterior aspect of the chest while standing behind the patient. There may even be slight retraction without disease of the apex. This I have proved by radiograms. The movements of the Adam's apple should be noted, as de la Camp[15] has observed that in some cases with enlarged thoracic glands, it is stationary instead of descending with inspiration.

Palpation. It would seem that palpation affords us the most reliable means of differentiating between active and inactive gland disease. Tenderness over the manubrium sterni and at the sterno-

[14] Loc. cit.

[15] Ergebnis. d. inn. Med. u. Kinderheil, 1908, i, 556.

costal junction is suggestive of bronchial adenopathy, but these tender areas occur less frequently than tenderness over the spines of the upper thoracic vertebræ. This was first described by Petruschky[16] some years ago, and other clinicians have since attested its value. It is best elicited as follows:

The examiner is seated, and the child with arms crossed, stands sideways between his knees, in order that the expression of the face may be noted. Then, without explaining what is to be done, firm pressure is made over the vertebral spines. The change of facial expression is the best indication of spinalgia, though in some instances sudden approximation of the scapulæ takes place when the tender spines are palpated. No tenderness being elicited by this means, it is then permissible to ask if all the spines feel just alike, and a repetition of the test may reveal a less marked tenderness. One should make firm pressure of the vertebral spines, going from above downward, and marking any tender spots; then begin in the lumbar region and go upward. The tenderness at times is extremely acute. It seems to be most often present over the fifth thoracic vertebra, though it is not uncommon to find spinalgia over several of the upper thoracic spines.

Petruschky does not explain what it is due to. He suggests that it may be caused by a periosteal irritation, the result of disturbances of circulation, or to the presence of minute tuberculous foci in the spinal column.

One should remember that MacKenzie has noted tenderness over the upper four thoracic spines in diseases of the heart, and over the fourth, fifth, sixth, seventh, eighth and thoracic vertebræ in stomach lesions. He, too, is unable to explain it. Inasmuch as all these viscera are supplied by the vagus, it would seem as though it were a reflex pain, but just how it takes place is not clear. The spinal tenderness of hysteria and neurasthenia is not limited to the upper thoracic vertebræ.

It is very likely that the backache in the interscapular region, so often complained of in early stages of tuberculosis, is due to tuberculous glands.

Petruschky reports 79 cases of spinalgia, 77 of which reacted to tuberculin. Advanced cases of tuberculosis do not present this phenomenon. The sign was present in 23 out of 58 children of my series; and 83 per cent. of them reacted. Ten adults recently examined, in whom there was a strong suspicion of an early lesion, had spinalgia; 80 per cent. of them reacted to tuberculin. In both children and adults with toxic symptoms this sign was rarely absent. It has been observed that both rest in bed and the therapeutic use of tuberculin cause a disappearance of the spinal tenderness.

[1] München. med. Woch., 1903, i, 364.

Neisser[17] has developed an ingenious method of palpation, which however, is not practical in children. He covers the end of a small stomach tube with a condom, and after introducing it, 25 c.c. to 26 c.c. from the teeth, inflates it through the tube. As the condom becomes distended, it presses on the glands, producing the characteristic pain between the shoulder blades. A number of observers have found this sign present in a large percentage of cases.

Percussion. The detection of dulness over the manubrium sternum has little diagnostic value. Up to about the sixth year the thymus give a dull note, as a rule. The left lobe, which extends higher than the right, can frequently be detected to the left of the sternum in the first interspace. According to Dautwiz, the thymus may persist to the thirteenth or fourteenth year. Then, too, so many other structures intervene between the sternum and bronchial glands that manubrial percussion is of little value. Bing[18] believes, however, that with *very light* percussion over the right half of the manubrium sterni, he can elicit slight dulness when there is glandular enlargement. Dulness over the sternum, or in the inner third of the first left interspace was present in 41 of our cases, 49 per cent. of whom reacted to tuberculin.

Parasternal dulness in the second and third interspaces, caused by enlarged hilum glands, was not sought for in this series of cases. It is possible that the dulness occasionally present in the second and third interspaces on the right, in the parasternal line with right apical disease, is caused by enlarged bronchial glands rather than by an increase of cardiac dulness (to which it is commonly attributed) from the retraction of the anterior border of the right lung caused by cicatrization at the apex. Though, as a rule, little is gained by percussion anteriorly, information of real value may be obtained by posterior percussion. Notwithstanding that spinal percussion was mentioned by Auenbrugger[19] it has never been extensively practiced. Advocated by Ewart,[20] and von Koranyi[21] and recently by De Costa,[22] it was de la Camp who made an extensive investigation of its value in the diagnosis of enlarged bronchial glands.

Without going into the elaborate studies of Koranyi, and Ewart, it is sufficient to state that a dull note is obtained normally over the upper four thoracic vertebræ, and that over the remaining

[17] Deut. Arch. f. klin. Med., 1905, No. 86, quoted by Nagel, Jahrb. f. Kinderh., 1908, lxviii, 46.

[18] According to Quain, quoted by Hall, Phila. Med. Jour., 1900, vi, 1059, manubrial dulness from bronchial glands can sometimes be obtained, if one percusses at the end of a forced expiration, with the head in dorsal flexion.

[19] Camac, Epoch-making Contributions to Medicine, Surgery, and the Allied Sciences, p. 125.

[20] Lancet, 1899, ii, 261.

[21] Handb. d. spec. Path. u. Therap., 1897, iv, 717, quoted by de la Camp (q. v.).

[22] Amer. Jour. Med. Sci., 1909, cxxviii, 815.

dorsal spines the note is a combination of osteal and pulmonary resonance. Dulness over the fifth or sixth dorsal spine is pathological, and is indicative of mediastinal tumor. Ewart states that the fifth spine is normally dull. This does not agree with my observations.

De la Camp attaches great importance to this dulness, and though he found it in only one-third of his cases of enlarged glands, it was in some instances the only sign of their presence. He suggests that percussion at the end of expiration may elicit slight dulness, otherwise not demonstrable. Nagel ascertained by experiments on cadavers, that 10 c.c. of wax injected anterior to the fifth dorsal vertebra would not produce dulness, but that 15 c.c. so injected would. He believes, that the dulness is not so much due to the glands per se as to the lessening of pulmonary resonance, from the pushing aside of the lung by the glands. Percussion of the vertebral spines was performed in only a small number of our cases. It was found at or below the fifth dorsal spines in 9 out of 40 children who were positive to tuberculin; 7 adults, all of whom had other signs of bronchial gland enlargement, had either vertebral or paravertebral dulness.

Krämer considers spinal percussion greatly inferior to paravertebral percussion. He is convinced by his investigations that if one have a true ear and percuss with sufficient delicacy, slight degrees of dulness can be detected whenever there is glandular enlargement. The dulness may or may not merge into apical dulness when the apex is involved. The dulness may be bilateral or unilateral, and when the former, the area of impaired resonance is usually unequal. Krämer attributes the dulness largely to the engorgement of the blood and lymph vessels, due to the glandular pressure. He believes, that the initial dose of tuberculin should be governed by the size of the area of dulness: the more extensive the dulness, the smaller should be the dose. As in all cases in which delicate percussion is employed, more trustworthy results are obtained if the eyes are closed and a dermographic pencil used. The sense of resistance is perhaps as significant as the impaired resonance. Paravertebral percussion was carried out in too few cases in this series to draw deductions, but in the cases in which it was detected other signs of bronchial glandular enlargement were present. The dulness at the right apex, which is sometimes present without disease of the lung, and attributed by Krönig to nasal obstruction from adenoids, is believed by Bing[23] to be due to enlarged bronchial glands, which tend to be arranged more in groups on the right than on the left side.

Auscultation. Like vertebral percussion, ausculation over the vertebral column has never been widely employed. A few years ago d'Espine[24] called attention to the fact that enlargement of

[23] Ugesk. f. Laeger., 1910, lxxii, 199.

[24] Bull. acad. de méd., 1907, lvii, 167.

the endothoracic glands gave auscultatory signs that were quite distinctive. In the normal individual the tracheal quality of the respiratory murmur ceases at the seventh cervical vertebra. When the bronchial glands are enlarged, however, the tracheal quality, somewhat diminished in intensity, is continued downward over the thoracic spines for a variable distance. In some instances the bronchial character is absent over the upper thoracic vertebræ, but present at the fifth, and possibly below. Except in the extremely emaciated one can place the bell of the stethoscope directly over the vertebræ. The character of the voice, especially the whispered voice, affords the most reliable evidence upon which to base a diagnosis of enlarged tracheobronchial glands. The words three thirty-three being repeated slowly, when glandular enlargement is present, bronchophony is quite distinct. If one listens with care, it will be observed in most cases that the final "e" of the last word "three" is heard for an appreciable time after the voice stops. Gray refers to it as the "postphonal quality." This is normally present over the trachea. It may be heard over the upper thoracic vertebræ, or only at the level of the fifth dorsal. I have heard it as low as the eighth dorsal.[25] The split syllable, referred to by Gray, was not observed.

As a rule, the bronchial quality is lost when the stethoscope is moved to either side of the vertebral column, but in some cases it is heard as distinctly at one side, rarely at both, as over the spines. In one instance, the whispered voice was so marked as to suggest a cavity at the apex of the left lower lobe. Flexing the head intensifies the bronchophony.

No infants are included in the cases herewith reported, but the sign is of equal, perhaps of greater value than in older children, as the signs of pulmonary involvement in infants are notoriously indefinite. In these little ones the character of the cry and of expiration must be substituted for the spoken words.

Vertebral bronchophony was present in 80 out of 168 children, and 66 per cent. of those in whom it was detected reacted to tuberculin. Those not giving the sign, gave 28 per cent. of positive reactions; 44 out of 80 frail children had vertebral bronchophony, and 70 per cent. of these were positive to tuberculin; 39 of the 80 in whom the sign was negative, reacted to tuberculin; 24 adults who had spinal bronchophony, gave 80 per cent. of positive reactions. I have recently had autopsy confirmation of the value of this sign. The necropsy upon a woman, aged sixty-one years, who had had vertebral bronchophony over the upper four cervical vertebræ and slightly prolonged expiration at the right apex posteriorly, revealed

[25] In an adult recently examined in whom the clinical diagnosis of enlarged bronchial glands was substantiated by a radiogram, vertebral bronchophony extended to the second lumbar vertebra.

a mass of partly caseous glands about an inch in diameter lying in the angle formed by the trachea and the right bronchus. The infratracheal glands were but slightly enlarged and the right apex showed no signs of infiltration. D'Espine reports several similar cases.

It is of interest to note that Laennec[26] detected this interscapular bronchophony. He says, "In persons, however, of delicate and feeble frame, particularly in *lean children* (italics supplied) there frequently exists in this (interscapular) situation a bronchophony very similar to the laryngophony, already noted." I was not able to detect the weakened inspiration at the apex, which Grancher[27] states is due to bronchial gland disease, and antedates the bronchophony, though faint breathing is commonly present in very early apical lesions. Bronchophony over the sternum was not observed.

Dilated veins over the anterior aspect of the thorax coexisted with vertebral bronchophony in 51 instances, and these cases gave 69 per cent. of positive reactions to tuberculin.

Some years ago Eustice Smith[28] called attention to the venous hum heard over the manubrium sterni, when the head is hyperextended, which disappears as the head assumes its normal position. He considered it a valuable sign of bronchial gland tuberculosis, and attributed it to the pressure upon the left innominate vein by the glands at the lower end of the trachea. Inasmuch as it is uncommon after the thirteenth year, and as one-half of the 78 of our cases who presented this sign failed to react to tuberculin, its value can be seriously questioned. It seems to be especially common in short necked children.

Rales. A little over one year ago, Miller[29] and Woodruff drew attention to the presence of crepitant rales in the fourth, fifth, and sixth interspaces at about the mid-clavicular line in many of the children suspected of having tuberculosis. A positive diagnosis of tuberculosis was made in 82 of the cases presenting fine rales near the nipple. Miller and Woodruff considered the rales very suggestive of a tuberculous lesion at that region.

Rales were present in 46 of 137 of our cases, and except in a very few instances were located in the region of the mid-clavicular line in the fourth and fifth interspaces; 65 per cent. of children presenting rales reacted to tuberculin, and 38 per cent. of children in whom rales were not detected reacted. Rales were present in 46 per cent. of children reacting to tuberculin, and in 22 per cent. of those who did not react.

As only one examination was made in over half of the cases, the observation has less value, as the rales may be heard at one

[26] Camac, op. cit., p. 178.

[27] Bull. acad. de méd., 1906, lxx, No. 36. Abstract, Jour. Amer. Med. Assoc., 1906, xlvii, 2041.

[28] Wasting Diseases of Childhood, London, 1899, p. 309.

[29] Jour. Amer. Med. Assoc., 1909, lii, 1016.

examination, and not at another. They seem to be as common in the robust as in the frail child. These rales may indicate a tuberculous focus at that point; but the fact that it does not tend to spread, and later give definite signs of tuberculous infiltration, nor leave postmortem evidence of a primary disease at that point, is against this supposition.[30]

RADIOGRAPHY. The *x*-rays are very valuable in the diagnosis of enlarged glands. Their use requires, however, perfect technique and a large experience in this particular line of work before they are of value. De la Camp considers it the most difficult of *x*-ray work.

Through the courtesy of Dr. Arthur C. Heublein, I have been able to compare the physical signs with the *x*-ray plate in 50 cases. Calcified glands are recognized by their sharp, clear-cut shadow. Cheesy glands, according to Nagel, give a shadow of less density, and with less clearly marked boundaries. The earliest evidence of tuberculosis of the bronchial glands seems to be a shadow that extends from about the anterior tip of the third rib on the right upward to the first rib. This shadow extends to the right of the vertebræ for a variable distance. It "bulges," as it were, into the region normally occupied by the inner aspect of the right lung (Figs. 2, 3, 4, 5, 6). In Fig. 2 the glands can be seen extending well up to the apex. Since making this observation, I find that Sluka[31] has arrived at identically the same conclusion. In very young infants he found that the lung adjacent to the glands was practically always involved. He states that the pulmonary lesion may heal so thoroughly that no pathological lesion can be found at autopsy, though the lesion in the glands persists. Krämer attributes this "eaten out" appearance, sometimes observed at the border of the shadow, to the engorgement of lymph and bloodvessels. Swollen, but not cheesy glands, Nagel states, are less clearly or not at all recognizable.

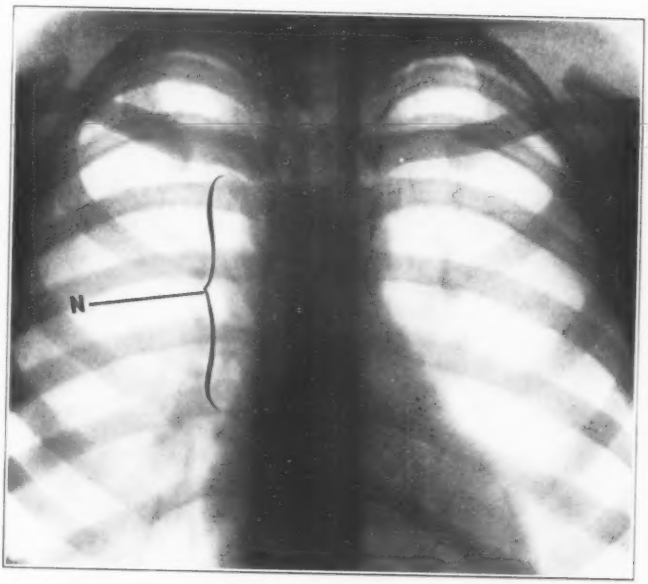
Recently I had an opportunity apparently to confirm this. A patient presenting a large mass of cervical glands, entered the Hartford Hospital for operation. A gland fully an inch in diameter, situated nearly behind the left clavicle, failed to cast a shadow, though the bronchial glands could be distinctly seen. At the operation it was found that the gland behind the clavicle was not caseous. It is possible that the dense muscles in which the gland was imbedded, obscured its shadow. A chain of glands can often be seen along the right border of the heart. The glands about the trachea give a more diffused shadow than the hilum glands.

Dr. Heublein obtained the best results when the anterior aspects of the chest was in contact with the plate. The large infratracheal

[30] In the discussion of this paper Dr. John H. Lowman said that he attributed these rales to the congestion of the lymph vessels from the presence of the enlarged bronchial glands.

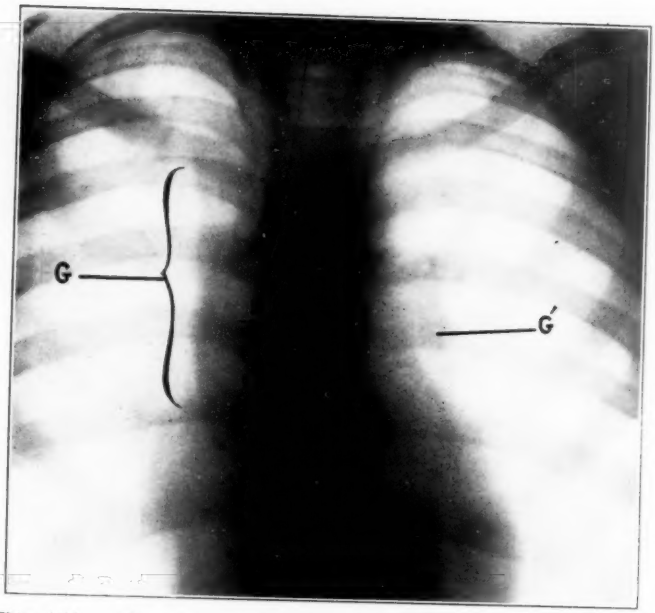
[31] Wien. klin. Woch., 1910, xxiii, 156.

FIG 1



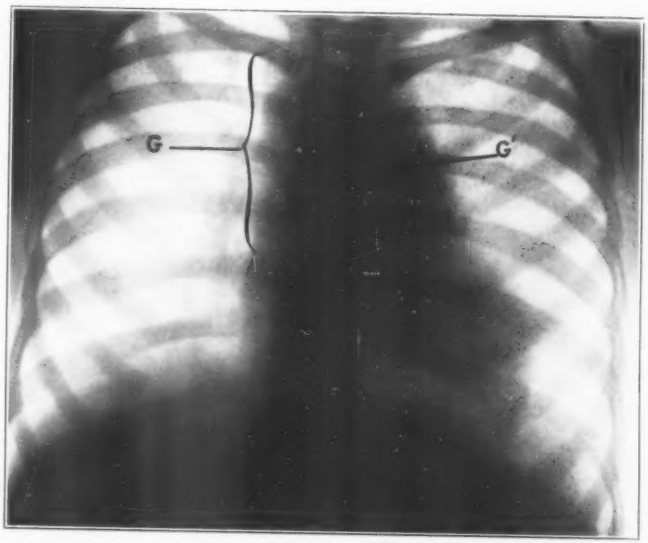
This child, aged twelve years, had none of the signs of bronchial gland enlargement, and was twice negative to the tuberculin skin test. The normal contour of the root of the right lung is shown at N. Compare this with the shadow of the bronchial glands in the succeeding pictures. In this case, as in all the others, the physical signs were recorded before the *x*-ray picture was taken.

FIG. 2



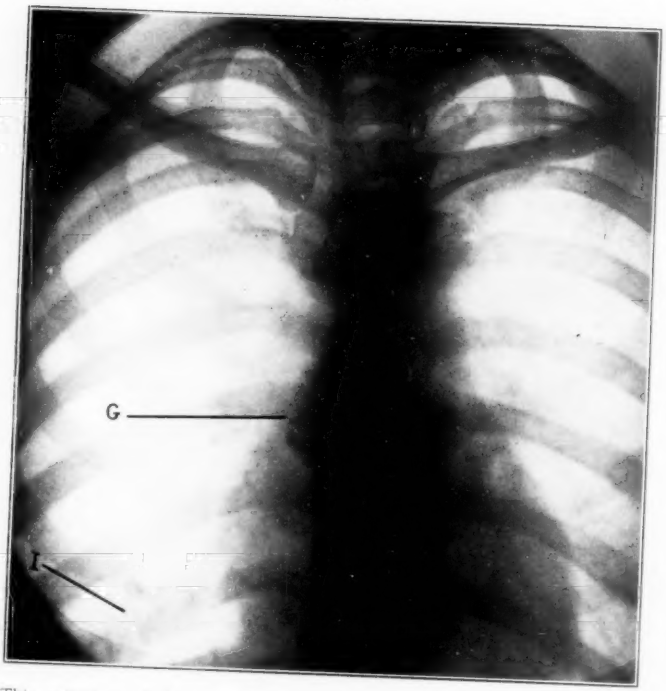
This child, aged seven years, had some retraction and diminished expansion at the right apex. Spinalgia was present over the upper thoracic vertebræ and the fifth thoracic spine was dull on percussion. There was no vertebral bronchophony. The tuberculin test was negative; repeated in six months with the same result.

FIG. 3



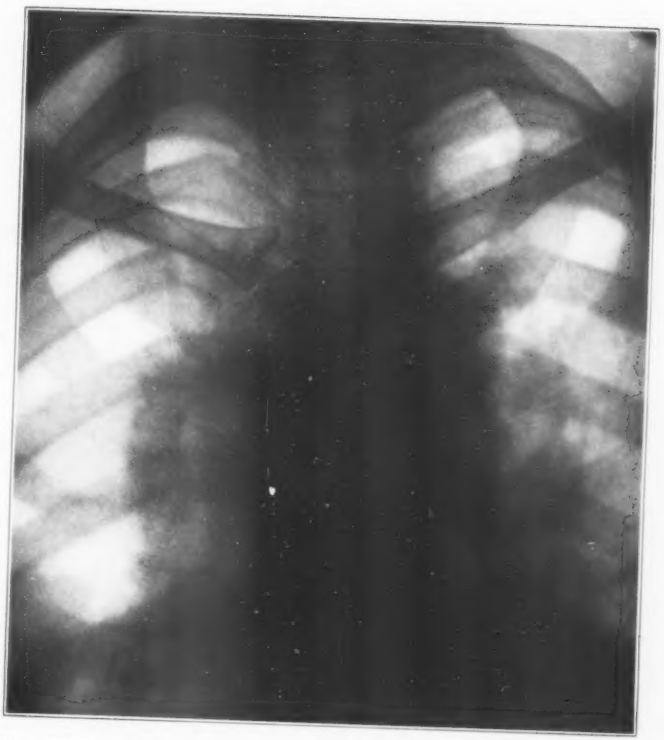
This child, aged eleven years, had some retraction below the right clavicle. Neither spinalgia nor vertebral dulness was present. Vertebral bronchophony was noted and the tuberculin test was positive. The glands extending upward along the right side of the trachea can be seen at G, and a gland above the left bronchus is indicated at G′.

FIG. 4



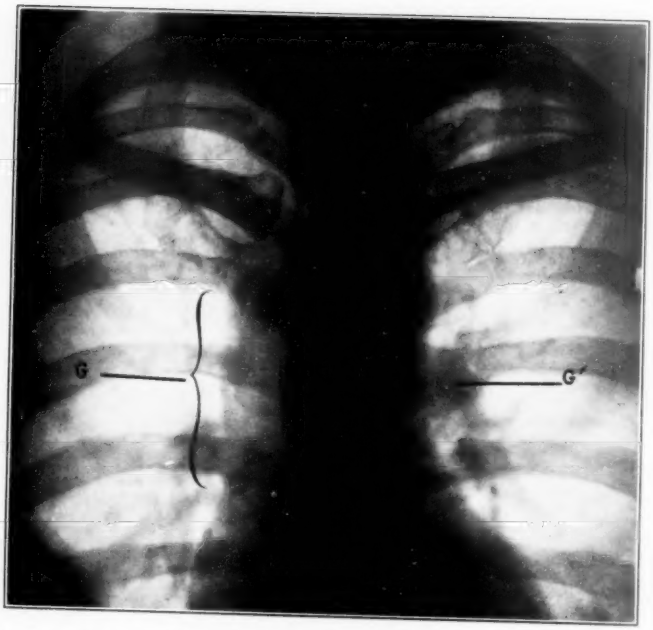
This patient, aged thirty-two years, showed slight lagging and muscle spasm at the left apex. Tenderness was present above and below the right clavicle and in the fifth right interspace in the parasternal line. Spinalgia was exquisite. There were both vertebral and paravertebral dulness (bilateral). Bronchophony over the vertebræ was present, but not marked. There was slight dulness at the apices, where a few fine rales were heard: rales also in fifth right space near the sternum. The *x*-ray plate showed deficient aeration at both apices and slight mottling at the right. An enlarged bronchial gland (presumably the infratracheal group) is indicated at G, while at I there is a small area of infiltration.

FIG. 5



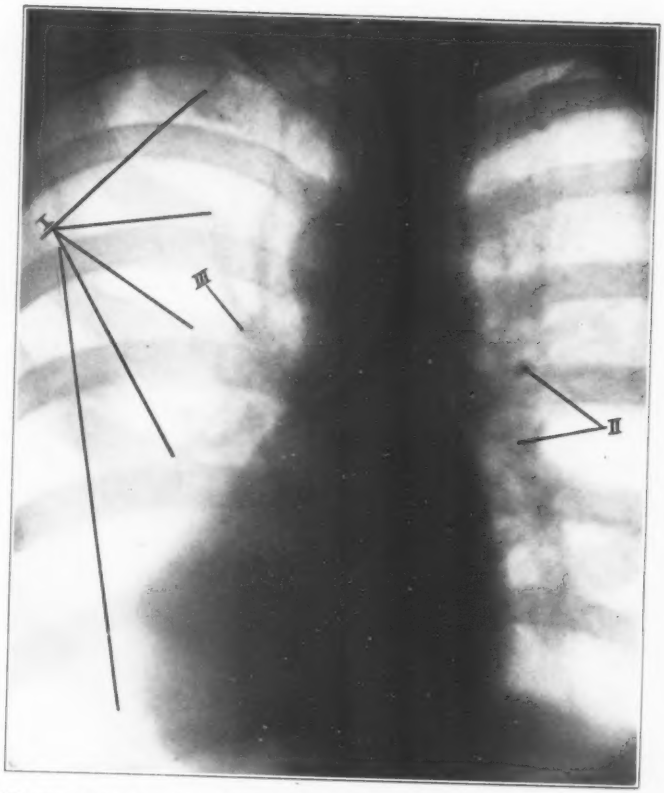
This patient, aged forty-nine years, had the usual signs of slight infiltration at the right apex. Vertebral and paravertebral dulness and vertebral bronchophony were present. Twice negative to the skin test and subsequently positive to the subcutaneous test. The patient has since died. The *x*-ray plate showed deficient aëration at the right apex and a large mediastinal tumor.

FIG. 6



In this patient, aged forty years, the signs were those of early infiltration at both apices and along the anterior borders of the lungs. Spinalgia was present, and there was slight dulness to the right of the fourth and fifth thoracic vertebræ. Vertebral bronchophony was present from the fifth to the eighth thoracic spines. It extended somewhat to the left. Individual tubercles can be seen above the right clavicle. The increase in thickening at the root of the lung, due to bronchial glands, is indicated by G, while a hilum gland can be seen at G'.

FIG. 7



Woman, aged forty-five years, with suspicious signs at the left apex and a positive tuberculin reaction. The apices are not shown in this picture, but in a subsequent one early infiltration at the left was apparent. Calcification of the costal cartilages is indicated by I; a small hilum gland at II, and extending upward from the gland at III, one sees streaks extending up to the left apex. These are probably thickened lymphatics, and indicate the path of infection from bronchial gland to the apex.

glands lie posterior to the heart and do not cast a shadow in most cases, though it is not uncommon to see small glands through the heart, near its periphery. Fig. 4, however, shows this infratracheal gland very distinctly opposite the second intercostal space on the right.

Quite commonly one sees an enlarged gland close to a bronchus, which is seen in cross sections. Fig. 7 seems to show the line of infection along the lymphatics from a hilum gland to the apex, which Sturtz[32] has described. The enlarged glands can be readily seen with the fluoroscope, and their appearance has been described by Minor.[33] An *x*-ray plate, however, allows more careful study, and is a permanent record.

Inasmuch as tuberculosis is not the only disease that causes enlargement of the endothoracic glands, it is desirable to substantiate the diagnosis by the use of tuberculin. The skin test of von Pirquet is the one of choice in children, bearing in mind, however, that occasionally a reaction will be obtained from the subcutaneous test when the skin test is negative. The intensification of pressure symptoms following the subcutaneous injection is indicative of a focal reaction in the glands.

Conclusions. 1. Tuberculosis of the bronchial glands often exists as a distinct clinical entity, capable of diagnosis.

2. While the diagnosis is more readily made in children, it can frequently be made in adults.

3. The presence of dilated veins over the anterior aspect of the chest, spinalgia, interscapular or vertebral dulness, and vertebral bronchophony speak strongly for enlarged bronchial glands, the tuberculous nature of which is practically assured when in addition to the above the individual is under weight and has a paroxysmal cough and the symptoms of the tuberculous toxemia.

4. The recognition of the disease, while it is still limited to the bronchial glands, is of the utmost importance, as we know the most salutary results of tuberculin therapy are obtained in glandular tuberculosis.

[32] Proc. Fourth Congress Tuberculosis Specialists, quoted by Wolff and Eisner. Ophth. and Cutan., diag. of Tuberculosis, London, 1908, p. 172.

[33] New York Med. Jour., 1910, xci, 573.

PELLAGRA, WITH A REPORT OF TWO CASES WITH NECROPSY.[1]

By Paul V. Anderson, M.D.,
OF MORGANTON, N. C.,
AND
William G. Spiller, M.D.,
OF PHILADELPHIA.

Clinical Report by Dr. Anderson. Case I.—Mrs. D. E., white, aged twenty-two years. The family history is negative except for the fact that one brother died with pulmonary tuberculosis. The patient had the diseases common to childhood. Catamenia occurred at sixteen, and was regular, but has been absent since March, 1909. She has always been in good health until four years ago, when she began to have gastric disturbance. This began in the spring, but became less severe during the fall. The following year, in March, her gastric symptoms returned and she had a persistent diarrhœa, with several watery stools a day. Later, the dorsal aspect of her hands and wrists became reddened and the palms roughened. There was a reddened patch on the neck, at the root of the hair, and on the nose and at the corners of the mouth. The patient states concerning her hands: "They were red, itched, and were sore, and looked just like they had been dipped in hot water and scalded." The elbows were also red and roughened. The mouth and tongue were sore. Salivation was profuse. Hunger and thirst were excessive, and there was considerable loss of weight. She was able to do her housework, however, and was not confined to bed. After a short time the skin of the hands and of the patches on the nose and neck peeled off. The diarrhœa gradually grew better, and in the fall the patient gained flesh and seemed to be in fairly good health. In March of the following year the eruption again appeared as before, accompanied by the symptoms previously enumerated, but they were more severe, especially the diarrhœa, and the patient became so ill that she was confined to her bed. In August, 1909, she was taken to a general hospital, where she remained for a month. When she left the hospital she was better; the skin of her hands had again peeled, but the hands were roughened. She was unable to take up her housework, however, on account of weakness and nervousness. She also was not able to walk well, and at times felt dizzy and blind.

The patient became disturbed mentally in January, 1910, and was admitted to the Western North Carolina Hospital for the Insane February 27, 1910. The duration of the mental trouble

[1] Read at a meeting of the College of Physicians of Philadelphia, December 7, 1910.

before admission was one month. Prior to admission she was sleepless, restless, apprehensive of bodily harm when left alone, silly in speech and behavior, and had hallucinations of sight. At one time she threatened suicide. The physician who committed the patient made a diagnosis of "neurasthenia bordering on melancholia." (This diagnosis has frequently been made in cases which later proved to be pellagra.)

Since the patient has been in the hospital she has been emotionally unstable, weeping without the slightest cause, and occasionally smiling in a wan way. As a rule, however, she has an anxious, apprehensive expression on her face. She says that her blood and nerves are wrong; that her uterus and stomach are terribly diseased. She complains of excessive pains in her ankles and knees. She says, also, that her back is weak and causes her pain. There is marked salivation—in fact, so great is this that her pillow is frequently wet by it. Her appetite is ravenous and her thirst is excessive. Her mouth and tongue are sore and she complains of a burning sensation in the œsophagus and stomach. She is constantly complaining of these symptoms and begs the doctor to give her medicine for them, as long as he is in sight. She also begs him pitifully not to let her die. In answering questions she is more or less confused, at times, and her memory is defective. She speaks in a monotonous voice, never varying the tone. She walks with difficulty, but for a few days after admission she was able to walk from her ward to the porch without assistance.

Physical Examination, March 19, 1910: The patient (Fig. 1) is well developed, but very much emaciated. She has an anxious, agitated expression on her face and cries and moans almost constantly, bewailing her fate. At times she has a mask-like expression of the face. There are no asymmetries of face. The ears are well formed; lobules attached. The pupils are regular; contracted; react sluggishly to light and to accommodation. There is no nystagmus and no strabismus. The conjunctivæ are sensitive to touch. The tongue is protruded straight, but marked coarse tremors are present. The edges of the tongue are reddened and the central part is covered with a brownish coating. Salivation is present to a marked degree. There is a reddish-brown discoloration extending from the base of the neck upward to the hair-line, and there is also a reddish purplish discoloration on the dorsal aspect of the hands, extending an inch or two above the wrist (Figs. 2 and 3). This is symmetrical on each side. Nothing abnormal is noticed about the palms. The normal lines of the skin on the dorsal aspect of the hands seem to be lacking. The chest is symmetrical, more or less flattened, and the subclavicular fossæ show a more or less marked depression on each side. No abnormality can be determined by percussion, but auscultation reveals harsh breath sounds at the left apex. The heart is apparently normal in size and no murmurs can be heard. The

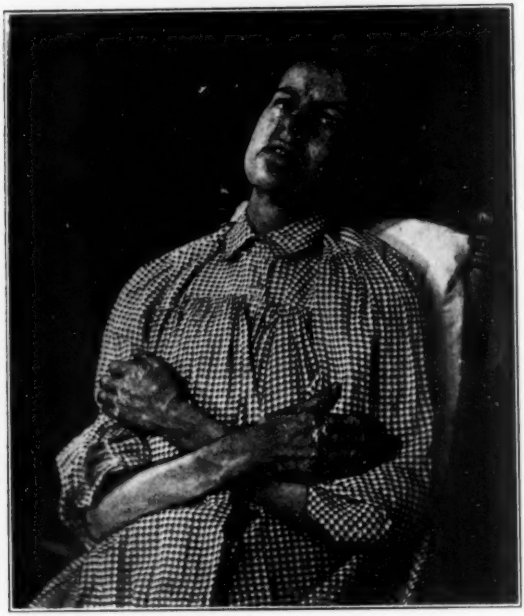


FIG. 1.—Photograph of the patient (Case I), showing the retraction of the head.

FIG. 2.—Photograph of the hands, showing the pellagrous eczema on the dorsal surfaces

FIG. 3.—Photograph of the right hand alone, showing the eczema more distinctly.

abdomen is slightly distended. No masses can be felt. The abdominal organs are apparently normal. The liver is contracted. It extends from the fourth interspace to the seventh rib in the midaxillary line. The abdominal reflexes are absent. The plantar reflexes are greatly exaggerated. The Babinski reflex and ankle clonus are not obtained. The patellar reflex is greatly exaggerated. The patient complains of excessive pain when she is tested for ankle clonus. There is a marked, coarse tremor of the head, and movements of the hands and arms are more or less athetoid or ataxic. There is marked incoördination in the upper extremities, the patient being unable to place her hand on her nose with the eyes shut. The station is poor; the patient shows a tendency to sway and would doubtless fall if she were not caught when the eyes are closed. The gait is more or less spastic and uncertain. She cannot walk without assistance, and walks with the feet spread wide apart. Apparently she has difficulty in recognizing the difference in the heat and cold tests on the feet and legs, but she could tell the difference when the tests were applied to her abdomen. The test for touch and pain could not be carried out on account of the patient's failure to cooperate. There are many coarse tremors of the hands when they are extended.

Smell: Turpentine; castor oil; ammonia, not recognized; camphor, correct.

Taste: Sour, correct; salt, correct; bitter, sour; sweet, not recognized.

March 30, 1910. Numerous bullæ have developed on the dorsal aspect of patient's hands, and in places these have broken, leaving sores. There is a marked reddening all over the dorsal aspect of the hands. There is also marked scaliness with excessive redness of the forehead, on the nose, and on the back of the neck. The elbows are very much roughened. The patient is quite emotionally unstable. She is fearful that she will die, and cries a great deal of the time. She almost invariably has an anxious, set expression on her face, and although, at times, she laughs in a silly way, her expression is more or less fixed all the time. There is marked redness of the mucous membrane of the mouth; the tongue is reddened and fissured, and there is an ulcer at each corner of the mouth. There is also marked salivation. She walks with difficulty—in fact, she cannot walk alone at all. The patellar reflex is greatly exaggerated. She eats ravenously and is excessively thirsty. She also, at times, has slight opisthotonos, and complains of excessive pains in her legs. She also states that her hands burn terribly. Her bowels are constipated. She sleeps scarcely at all.

April 15, 1910. The patient is growing much weaker. She has a persistent diarrhœa, with several watery, foul-smelling stools a day. She is excessively nervous and her muscles twitch a great deal. This condition exists even in her sleep. She is greatly depressed

and is apprehensive that something terrible is going to happen to her. She cries a great deal and begs constantly for food and water. The deep reflexes are still greatly exaggerated. The opisthotonos is increasing in severity. She cannot walk at all and has to be carried to her chair on the porch.

May 1, 1910. The patient is confined to bed. There are marked clonic convulsive movements of her legs and arms, and twitchings of her whole body; in fact, she is scarcely still a minute. The slightest touch produces a marked convulsive movement. She is still excessively thirsty and eats ravenously. She has a persistent diarrhœa, with numerous watery, foul-smelling stools each day. The dorsal aspect of her hands presents a reddish, purplish appearance. The epidermis is coming away in scales. There is also a scaliness about the corners of the mouth, on her nose and forehead, on the back of her neck, and on her elbows.

May 8, 1910. The patient is much weaker. Speech is thick and she expresses herself with difficulty. At times she is delirious. There is a very marked opisthotonos and there are clonic convulsive movements of the arms and legs. At times there are tonic spasms, and marked muscular twitchings are present most of the time. The mouth is excessively reddened, with saliva drooling from it, and the tongue is beefy in appearance. The deep reflexes are still greatly exaggerated, but are not so much exaggerated as they were for a time.

May 9, 1910. The patient died today.

March 1, 1910. Urinalysis: Straw-colored, flocculent precipitate; slightly acid; 1010; trace of albumin; no sugar; no casts; many urates; a few epithelial cells.

March 3, 1910. Examination of sputum for tubercle bacilli, positive.

April 23, 1910. Urinalysis: Light amber; clear; acid; 1013; no albumin; no sugar; no casts; many epithelial cells.

April 23, 1910. Feces examined—no hookworm ova found.

Case II.—E. A. H. (under the care of Dr. Jas. K. Hall. Notes by Dr. Hall).

This man was admitted to the Western North Carolina Insane Asylum in 1888, at the age of forty, from a county in the extreme western portion of the State. He had been married for ten years and had a wife and two children. He was a gunsmith and cabinet-maker, but he had no education and was without property. For ten years or more before admission he had been wandering about over the neighborhood, had done little work, and for perhaps more than ten years he had been looked upon as mentally abnormal. At times he was mute for days; sometimes he was extremely irritable, had visual hallucinations, and he occasionally threatened violence to members of his family and his neighbors. He attempted suicide, and pending admission to the hospital he was kept in the county jail for several weeks.

When admitted to the hospital the patient was a quiet dement, in rather frail physical health. Two years after admission he had a severe attack of influenza, but he made a good recovery, and six years later he had measles. When able, he worked on the farm, but there was a loud, systolic murmur, and at times the feet became so swollen that confinement to bed was necessary for a short time. In the latter part of 1903 he was transferred from the main building of the hospital to the colony, where the patients all engage in outdoor work, and this man found employment in breaking stones with a hammer, for macadamizing the roads. The note was made that his physical health during the winter of 1903-04 was first-rate, but a line in his history, dated April 1, 1904, states that he has an eczema-like eruption on his hands, and for that reason he is left indoors. On June 16, 1904, it was noted that he had been in bed for several days because of disturbances of cardiac compensation; that he had been unusually depressed and ate little, but the condition of the hands had improved. In the early part of October he was said to be very weak, emaciated, and there were bedsores. The physical condition had improved by the spring of 1905, and he was again able to be out of bed. For the following two years he remained in the hospital building, doing a little work in the ward, and it is presumed that his physical health was fairly good, inasmuch as no mention is made of illness in the history. On May 15, 1907, the following note was made: "This patient was transferred from the hospital building to the colony about April 1; he busies himself in helping to keep one of the buildings in order, but he is profoundly demented; and the scope of his intellectual activity is extremely circumscribed. It is impossible to induce him to raise his voice and he speaks always in a whisper. His general health is not robust, but apparently fairly good." The old man remained at this colony until September 24, 1909, when he was again placed in a hospital ward because of failing physical health. At that time the backs of the hands looked as if they had been badly sunburned. The bowels were loose, the appetite was failing, and he was losing in weight. The gait was uncertain and tottering; the movements of the upper extremities were athetoid; the protruded tongue was tremulous, and the deep reflexes were exaggerated. Ankle clonus and Babinski's sign were absent. No doubt was felt that the condition was due to pellagra.

The decline was steady and progressive. The diet was restricted principally to milk, and although sufficient nourishment was taken, emaciation increased. Dementia was so far advanced that no intelligent statements could be elicited from the patient. Diarrhœa was troublesome, and all control of the movements was soon lost. Tonics were given and efforts were made to control the diarrhœa, but without avail. As the emaciation increased, bedsores developed. The bed was soiled every few minutes. The bowel

movements were a dark, grayish liquid, and peculiarly offensive. The lower extremities became drawn, and efforts to extend them seemed to cause great pain. There were almost constant, apparently purposeless, movements of the upper extremities, extremely awkward and incoördinate. He was extremely restless and his behavior induced the belief that he was annoyed by visual hallucinations. As the disease progressed, the muscular disturbance became more marked. In addition to the gross movements of the upper extremities, there were spasmodic movements of the fingers. The eyelids frequently moved rapidly. At times there were clonic, convulsive-like movements of the whole body, of momentary duration. Not infrequently an unusually loud inspiratory grunt could be heard, due probably to spasmodic contraction of the diaphragm and other inspiratory muscles. Firm pressure on the skin seemed not to produce pain, but the gentlest tap on any part of the surface caused generalized, clonic muscular spasm. Pupillary abnormality was not noted. The temperature was never much above normal and in the last days it was slightly subnormal. The pulse became quite small and rapid. Urinalysis showed a trace of albumin and a few hyaline casts. Repeated differential blood counts invariably showed an increase in the number of large lymphocytes. One count gave the following result: Polymorphonuclear neutrophiles, 52.75 per cent.; eosinophiles, 2.5 per cent.; large lymphocytes, 22.75 per cent.; small lymphocytes, 22 per cent.

Death occurred January 3, 1910.

The necropsy showed nothing unusual. Emaciation was extreme; the muscles were quite red; the lungs and pleuræ were normal; the heart was somewhat enlarged, and the mitral leaflets were gnarled. The spleen was small, dark, and tough; the liver was about normal in size and consistence, and the intestinal wall was thin and pale. The kidneys were small; the capsule was adherent; the organs were tough and the cortex shrunken.

Remarks. It will be observed that this man was an inmate of the institution for twenty-two years before his death. During this time he had lived a sheltered, protected life, in a climate noted for its healthfulness. He was probably the first patient in the hospital to develop pellagra. It is scarcely to be doubted that the soreness of the backs of his hands noted in 1904 was the dermatitis of pellagra. For the following five years there were apparently no symptoms of the disease. The final attack developed late in the summer. For more than two years before his last illness he lived in a colony with seventy-five other patients, almost a mile from the hospital, and during this time he was not near any pellagrous patients. His diet and his hygienic conditions were exactly those of a thousand other patients. Contagion was not possible. If the food and surrounding conditions caused the disease it seems strange that he alone should have been affected.

THE PATHOLOGY OF PELLAGRA BY DR. SPILLER. The number of clinical cases of pellagra reported in this country has become quite large since the disease was first recognized here a few years ago. So far as I know, no report of a necropsy in any of these cases has been published, and it is important to determine whether the pathology of the American cases conforms to that of the European or not. It is presumable, however, as the symptoms of the disease are essentially the same in our country and in foreign lands, that the pathology will not vary greatly. Pathological reports are found chiefly in the Italian language, and the only one in English known to me is by Batten, who studied the spinal cord in three cases described by Sandwich.[2] Batten found degeneration of the posterior columns in two cases, but the third case showed practically no change. He believed the sclerosis of the posterior columns to be of root origin. The process was a chronic one, as shown by the fact that the posterior columns were extensively sclerosed and no degeneration was revealed by the Marchi stain. This report by Batten is especially interesting in that it seems to show that the degeneration in the spinal cord was not rapidly progressive, otherwise changes would have been detected by the Marchi stain.

Tanzi,[3] in the recent English translation of his text-book, says that Tonnini, in 1883–1884, was the first to associate the spinal phenomena of pellagra with primary spinal lesions (cellular pigmentation) of the anterior and posterior horns of the spinal cord. In cases of long standing there may be general or local œdema of the brain, hyperemia, and thickening of the meninges, chronic internal hydrocephalus, and sometimes softening of the cerebral substance and hemorrhages. Accumulations of pigment are also to be observed in the cells of the spinal cord and in those of the sympathetic ganglia. The spinal cord at an early stage exhibits traces of systemic degeneration of combined type, that is, lesions of the posterior columns and of the crossed pyramidal tracts, as has been shown by Tuczek and Belmondo. There is also very often small-cell infiltration of the spinal meninges. In the acute cases the nerve cells of the cerebral cortex are swollen; in some instances the nucleus is displaced to the periphery, the processes are atrophic, and there is central chromatolysis.

Babés and Sion,[4] in their monograph on pellagra, state that in most cases of this disease nothing peculiar was found in the spinal roots, but in other cases notable degenerative changes were found in the posterior roots. In the spinal cord were pronounced pigmentation of the nerve cells of the anterior and posterior horns, and often combined sclerosis.

[2] Jour. of Path. and Bact., 1901, vii, 460.

[3] A Text-book of Mental Diseases, translated by W. Ford Robertson and T. C. Mackenzie, Rebman Co., New York.

[4] Nothnagel's System,

Babés and Sion, in their own cases, found the posterior roots often degenerated, and the degeneration of the posterior columns was secondary to this, but differed from that of tabes in the less involvement of Lissauer's zone and of the anterior root zone, unless the pellagrous changes in the posterior columns were far advanced, when these zones also were degenerated. The cells of Clarke's column were always affected. The cells of the anterior horns were degenerated, as were also the cells of the cerebral cortex. They regard the changes as produced by some poison.

Lukács and Fabinyi,[5] in a recent paper, state that system degeneration of the spinal cord is rare in pellagra. The cerebral meninges in their cases were intact, but in one case the spinal pia was a little thickened in places and infiltrated with cells. They found no unusual arteriosclerosis, although Marianis records arteriosclerosis as constant in pellagra. The medullated fibers of the brain, cerebellum, and medulla oblongata were normal (Marchi's method, however, was not used). In two cases the spinal tracts were intact, in a third the columns of Goll were degenerated from the pyramidal decussation to the upper thoracic cord. Many cells of the posterior horns were degenerated from the cervical swelling downward, but less intensely in lower levels, and this cellular degeneration is supposed by the writers to have been the cause of the degeneration of the columns of Goll. Degeneration of these columns, they state, is very common in pellagra, and differs from that of tabes in that it is seen only in the cervical region, and in that Lissauer's zone is intact. The cells of Clarke's column were greatly degenerated. Nuclei of sensory nerves in the medulla oblongata and pons were scarcely altered, while motor nuclei here were intensely affected. The cells of the anterior horns were greatly degenerated, especially in the cervical region, and showed granular chromophilic elements at the periphery, and a homogeneous centre, with absence of nucleus. The cells of the brain cortex were likewise affected, and especially those in the central convolutions. The lesions, they state, correspond to those of ergotin, mercury, or aconite poisoning, and pellagra is a disease of toxic nature.

Parhon and Papinian[6] seem to be the only investigators who have studied the neurofibrils in pellagra. They found the alterations less intense in the small cortical cells of the brain, and most intense in the Betz cells. The neurofibrils stained less deeply, were thinner, even fragmented. In some small pyramid cells they were not altered. In the large cells the neurofibrils were almost completely absent; in some they were preserved at the periphery and in the cell processes. Alterations were more intense in the cervical region than in the lumbo-sacral region, and were of the same kind as in the Betz cells.

[5] Allg. Zeitsch. f. Psych., 1908, lxv, 657.
[6] Comptes-rend. de la Soc. de biologie, 1905, lviii, 360.

Pathological Report of Case I (Case of Dr. Anderson). The nerve cells of the anterior horns of the lumbar region are intensely degenerated. The cell body is swollen, the nucleus is displaced to the periphery, the dendritic processes of many cells have disappeared, and the cell body shows intense chromatolysis, sometimes implicating the whole cell, sometimes leaving a zone of chromophilic elements at the periphery of the cell body. The cells of the anterior horns stained by the Bielschowsky silver stain show an absence or a fragmentation of the neurofibrils. There is no round-cell infiltration of the pia. The anterior and posterior roots appear to be

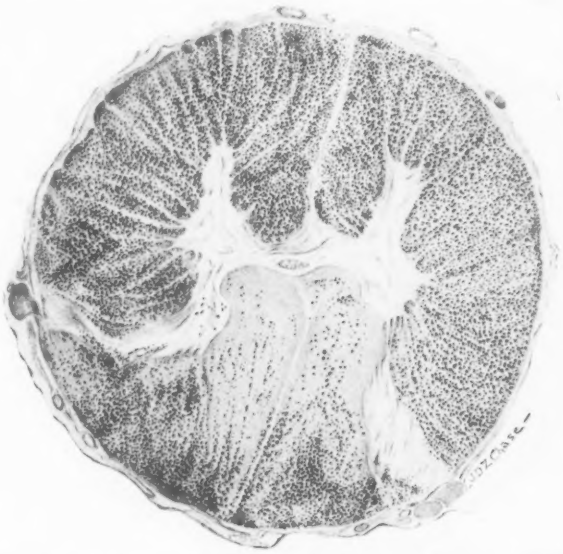


FIG. 4.—Drawing of a section of the thoracic cord from Case I, showing the diffuse degeneration of the posterior and lateral columns by the Marchi stain.

normal. Marchi's stain reveals diffuse degeneration in the posterior and antero-lateral columns, but less in the posterior columns near the gray matter. Numerous holes, from which nerve fibers have disappeared, are found in the antero-lateral columns. The pia is not thickened and the bloodvessels are not sclerotic. The thoracic cord is much distorted, probably as a result of injury at the necropsy. Here and there a swollen axis cylinder may be found in the antero-lateral columns. The cells of the anterior horns present the same condition as do those in the lumbar region. The cells of the columns of Clarke show similar changes. The degeneration of the posterior and antero-lateral columns by the Marchi

stain is pronounced (Fig. 4). Only the lowest part of the cervical cord was obtained. The anterior horn cells are in the same condition as are those in the lumbar region. The anterior and posterior roots of the eighth cervical and first thoracic segments, cut separately from the cord, appear to be normal. The capsules of the cells of the lumbar posterior ganglia show much proliferation of their lining cells, and the nerve cells are in part degenerated. Cells of the hypoglossus nucleus present a condition similar to that of the cells in the anterior horns, but of less intensity. The cells of Betz and the nerve cells of the deeper cortical layers in the paracentral lobules show changes similar to those of the anterior horn cells, that is, swelling, chromatolysis, and displacement of the nucleus (Fig. 5).

FIG. 5.—Drawing of nerve cells from the paracentral lobule and the anterior horns of the lumbar region. Cell of Betz from the paracentral lobule. One of the larger pyramidal cells of the deeper layers of the paracentral lobule. Cells of the anterior horn of the lumbar region. All these cells are much degenerated.

Pathological Report of Case II (Case of Dr. Hall). Only a small piece of the spinal cord was obtained, and this was from the thoracic region. The pia is distinctly thickened. Diffuse but slight proliferation is found in the lateral and posterior columns; the cells of the columns of Clarke are much swollen; the nucleus is eccentric, and the dendritic processes of some of the cells have been lost. The cells of the anterior horns are in a similar condition, but in some of these the chromophilic elements have disappeared from the periphery of the cell and are found nearer the nucleus; in others they persist only at the periphery. Spaces are found in the anterolateral columns from which nerve fibers have disappeared. The vessels of the cord are somewhat sclerotic. The Marchi stain shows

intense recent degeneration in the peripheral parts of the posterior columns, in the crossed pyramidal tracts and anterolateral columns, but less degeneration directly in front of the crossed pyramidal tracts and in the median and ventral portions of the posterior columns. The connective tissue of the sciatic nerve and the intima of the vessels in the nerve are proliferated. The nerve fibers of this nerve are normal.

It is evident from the brief abstracts of the foreign representative papers treating of the pathology of pellagra that there is a difference of opinion as regards the systemic or non-systemic appearance of the degeneration in the spinal cord. This is a matter of some importance, as a toxic degeneration is more likely to be non-systemic. In the two cases I have studied the degeneration was diffuse. Indeed, from my own experience I have come to believe that, with the exception of Friedreich's ataxia, there are few disorders causing a truly combined systemic disease of the spinal cord. The degeneration observed in anemia is not systemic, but in the cases I have studied (quite a large number), it has invariably been a diffuse process. It is more intense than that I have observed in pellagra, but not of an essentially different character. The nerve cells, however, are much more affected in pellagra than in anemia.

Writers seem to agree as to the pronounced degenerative changes in the cells of the anterior horns of the spinal cord and of the cortex of the cerebral hemispheres in pellagra. It appears from the first case reported in our paper that the moderate degeneration of the pyraminal tracts predominated clinically over the apparently more intense alteration of the cells of the anterior horns, so that exaggeration of patellar reflexes was present.

The view has been expressed (Long) that the thickening of bone about the intervertebral foramina, possibly more in the cervical region, may be a cause of the symptoms. Unfortunately, only the extreme lower part of the cord in the cervical region in Case I was obtained, but posterior and anterior roots from this region, cut separately, showed no distinct degeneration, even by the Marchi stain. Had these roots been implicated in the intervertebral thickening, degeneration would have been detected in them. I have found no indication of root degeneration in the thoracic or lumbosacral region, and the alteration of the posterior columns, at least below the eighth cervical segment, is clearly endogenous, as it is also in the piece of thoracic cord obtained in the second case.

I must conclude, so far as a study of these two cases permits, that pellagra does not always produce, if indeed it ever does produce, a truly systemic disease of the central nervous system; but that the degeneration is caused by some toxic or infectious substance affecting all parts of the cerebrospinal axis, producing cellular degeneration and diffuse degeneration of nerve fibers in the posterior and antero-

lateral columns. It is not difficult to explain the mental symptoms when cortical degeneration is so intense as may occur in pellagra, and as is seen in the brain I have studied; and the insanity of this disease seems to be of a toxic or infectious character.

The constant involuntary movements of the limbs described by Dr. Anderson, and present also in Dr. Hall's case, are suggestive of the similar movements in strychnine poisoning. In pellagra the degeneration of the cells of the anterior horns probably causing irritation in the muscles innervated by them, the impairment of cerebral inhibition produced by partial degeneration of the pyramidal tracts, and the incoördination produced by the partial degeneration of the posterior columns, the cerebellar tracts, and the cells of Clarke's columns, may all assist in causing involuntary and constant movement. It is sometimes forgotten that holding the hand still and extended is a very perfect form of movement, requiring the degree of tonicity in the flexors and extensors of the limb exactly sufficient to maintain this position.

The spinal pia was considerably thickened in the second case, which was of long duration, but the thickening of the pia was not observed in the first case.

THE INFLUENCE OF THE TRENDELENBURG POSITION ON THE QUANTITY OF URINE EXCRETED DURING ANESTHESIA.

By J. Wesley Bovée, M.D.,

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At the meeting of the American Gynecological Society in 1909, I presented a report of investigations made in my clinic in the Columbia Hospital for Women regarding the influence on renal activity from anesthesia by ether and by chloroform during surgical operations. I had not then thoroughly studied the variation due to the Trendelenburg position, but recorded my having been greatly impressed by it. Eight cases were therein noted as illustrative, and five cases had been especially studied. Having recently studied eight cases of ether anesthesia and eight of chloroform, I present herewith a report on those observations. I believe the results demonstrate that almost no urine is received in the bladder while the Trendelenburg position is being employed.

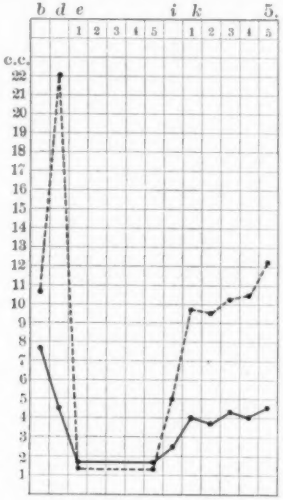
Technique. The quantity of urine for a twenty-four-hour period fairly remote from the operation was obtained. Care was used to secure this knowledge without such influences as undue excitement, abnormal intake of fluids, or catharsis. The usual diet was continued until about twelve to fourteen hours before operation.

During this twelve to fourteen hours liquid food in very limited quantities was given. The bowels were moved two or three times, the last by an enema, and nothing but the one anesthetic given. No morphine, atropine, or other drug was used, nor was salt solution in any manner employed. The bladder was emptied by catheter when the administration of the anesthetic was begun; when about to place the patient in the Trendelenburg position, the clamp was removed from the permanent catheter and the bladder emptied. At the end of each fifteen minutes of the time the patient was in this position the bladder was drained. This was facilitated by the fingers pressing, intraperitoneally, upon the bladder. The urine was drawn immediately after the patient was returned to the horizontal position, and at the end of each subsequent fifteen minute period during the administration of the anesthetic, as well as when the anesthetic was stopped. The head nurse of the operating room, Miss Pugh, was very accurate and greatly interested in this feature of the work. During the following two days the urine was carefully measured, usually every fifteen minutes of the first two hours, by means of the permanent catheter.

a	*b*	*c*	*d*	*e*	*f*	*g*	*h*	*i*	*k*				
				1 2 3 4					1	2	3	4	5
1	660	35	20	0 0	35	1	10	4	10	10	8	8	8
2	870	41	2	0 0 0 0	64	4	0	4	4	4	4	4	4
3	600	45	16	0 0	38	12	7	4	7	2.5	2.5	2.5	2.5
4	630	32	4	0 0 0	48	6	23	1.6	1	1	1	1	4
5	.....	26	22	0	19	4	15	1	1.6	2	2	2	8
6	1080	45	2	0	20	0	50	0	2	4	4	1	1
7	.....	85	24	0 0	40	8	26	0	2	2	8	8	4
8	690	35	12	0	15	1	15	4	4	4	5	5	5
Ave.	7.9	...	4.45	.....	...	1.9	...	2.3	4.0	3.7	4.3	4.0	4.5
1	840	38	44	0	23	8	30	2	40	40	40	40	40
2	1080	35	144	0	17	12	47	8	1.3	1.4	1.4	4	16
3	1110	41	4	0	29	0	24	7	6	6	6	6	5
4	840	35	32	1 0 0	50	0	16	0	0	0	0	0	0
5	480	40	20	.....	10	8	42	12	8	12	20	24	24
6	1230	33	8	0 0	39	6	21	1	16	12	8	2	4
7	870	50	24	0	29	8	7	8	8	7	8	8	8
8	1800	36	168	0 0	31	0	9	0	0	0	0	0	0
Ave.	10.7	...	22.0	.....	...	1.5	...	4.8	9.9	9.8	10.4	10.5	12.1

Explanation of the Table. The first series is ether anesthesia, and the second chloroform. The letter *a* signifies the case number; *b* the cubic centimeters of urine for twenty-four hours; *c* the number of minutes elapsed from the beginning of the administration of the anesthetic to the placing of the patient in the Trendelenburg position. The amount of urine excreted during that period is given in column *d*. The amount excreted during the employment of the Trendelenburg position is represented in fifteen minute periods under *e* 1, 2, 3, and 4; *f* records the exact number of minutes that position was used, while *g* represents the amount of urine drawn from the bladder when the patient's position was changed back to the hori-

zontal. The number of minutes the anesthetic was administered after using the Trendelenburg position is noted in column *h*, while the average amount excreted during each fifteen minutes of that period is found in column *i*; *k* 1, 2, 3, 4, and 5 represent urine quantities for the five periods of fifteen minutes each after the anesthetic was stopped. In every case the amount of urine became normal not later than twenty-four hours after operation.



The figures at the left of the chart indicate the quantity of urine for fifteen-minute periods, and the letters across the top of it refer to the corresponding columns in the table. The broken line represent the chloroform series, and the solid the ether series. It will be noted that in both series (ether 1.9 and chloroform 1.5) the current of urine in the Trendelenburg position suddenly comes down at the beginning to below 2 c.c. per fifteen minutes, and at once raises when the patient is taken out of that position.

In some of the cases in the table it will be noted that following the operation no urine was excreted during the hour and a quarter subsequent to the end of the operation. In each instance, at the end of twenty-four hours the quantity and quality were practically normal. In the chloroform series two patients excreted unusually large quantities during the early part of the anesthetic—one 144 c.c. in thirty-five minutes, and the other 168 c.c. in thirty-six minutes. These two cases extravagantly increase the average amount for fifteen minutes to 22 c.c., which is greatly in excess of the average (8.2) for the other six cases of the series. This gives a striking difference on the chart.

In the paper of 1909 I mentioned the marked lowering of the rate of urinary flow in the Trendelenburg position, estimating it at 32 per cent. In the series now submitted the decrease is for ether 58 per cent. and for chloroform 95 per cent. The decrease in the chloroform series is very much exaggerated, though in the six cases (omitting Cases II and VIII) of the series, in which the flow before using the Trendelenburg position was about normal, the decrease was 82 per cent. It might be said, therefore, that while the patient is in the Trendelenburg position the percentage of decrease in the excretion of urine is 58 per cent. in ether anesthesia and 82 per cent. in anesthesia by chloroform. That this great decrease is not, even in moderate degree, due to urine being retained in the renal pelvis is clear, for the rate of flow subsequent to changing the patient to the horizontal position is not suddenly greatly increased, being but slightly increased in the ether series, a little more in the chloroform series, and not for an hour and a quarter reaching a rate in excess of that of the period preceding the use of the Trendelenburg position. Nor can it be said that the bladder was not satisfactorily drained by the catheter, inasmuch as the fluctuations were always gradual and never sudden, except when changing to or from the Trendelenburg position.

If it can be concluded that the renal function is greatly lessened while the patient is in the Trendelenburg position, then the danger of that position is at once appreciated. In renal inefficiency, and cardiac and arterial lesions, it seems that the use of the Trendelenburg position introduces a special element of danger, and this less markedly when ether is used than when chloroform is employed as the anesthetic.

MULTIPLE ABSCESSES OF THE NASAL SUBMUCOSA IN A CASE OF LEUKEMIA.

By Joseph P. Tunis, A.B., M.D.,
FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA.

During April and May of 1909 I had the opportunity of examining, in the autopsy room of the Allgemeines Krankenhaus, in Vienna, the nares and accessory sinuses of ten adult heads in the clinical history of which leukemia had played an important part. In only one of these ten cases were multiple abscesses present. They gave the appearance of punctate hemorrhages over the entire nasal mucous membrane, more especially over the turbinate bones. This condition I believe is rarely met with, and therefore warrants detailed description. For permission to report these cases, as well as for the opportunity to study them, I am indebted to the courtesy

of Professor Hofrat Weichselbaum and Professor Stoerk. For additional notes on the clinical history I am indebted to Dr. Bartel. The history of this case is as follows:

Joseph G., a baker, aged forty-four years, married, was admitted to the General Hospital in Vienna on April 7, 1909, in the service of Professor Strumpell. The clinical diagnosis was diathesis hemorrhagica, with well-marked anemia, atrophy of the bone marrow, and lobular pneumonia. He died on April 16, that is, nine days later, and the autopsy was performed on the following day.

The patient's mother died at the age of seventy-six, from asthma and degenerative myocarditis. His father, at this time, was living and well. He had had no brothers or sisters. As a child he had scarlet fever and measles. At the age of fifteen he had a retropharyngeal abscess, and at the age of twenty-five a return of this abscess. At the age of twenty-one he had syphilis, and was treated in Professor Lang's clinic. At the age of thirty-five he married and has had no children. At the age of forty-three he had a severe attack of gastro-intestinal catarrh, with considerable colic. He states that at this time he vomited blood, and that his recovery was a tedious one. After an interval of several months there was a return of this gastro-intestinal trouble.

He had been a moderate drinker of alcoholic liquors. Since childhood he had often had slight hemorrhages from the oral mucous membrane, dyspnœa, and palpitation of the heart. His last illness began about three weeks before death with hemorrhages from the gums, epistaxis, and swelling of the cervical glands.

An examination of the blood on April 15 showed: (1) The red blood cells to be 1,368,000; hemoglobin, 20 to 25 per cent.; and the color index, 0.8 to 0.9, or a trifle less than normal. The rouleaux formation was diminished. There were no macrocytes, numerous microcytes, slight poikilocytosis, and a few faintly colored red cells. (2) Leukocytes, 3700; polymorphonuclear cells, 3 per cent.; lymphocytes, 45 per cent.; eosinophiles, 0.5 per cent.; Turck's irritation forms 2 per cent.; no mast cells. In addition, there were 29.5 per cent. of various cells, such as large mononuclear, marrow cells, and transitional forms.. (3) There were no blood platelets present; the coagulability was diminished.

The urine showed serum albumin absent; nucleo-albumin present; otherwise negative.

In the feces the chemical test for blood was positive.

Autopsy Notes. Acute lymphatic leukemia. Infiltration of the gums with gangrene of the upper jaw in the region of the incisors; leukemic infiltration of the nasal mucous membrane, with narrowing of the nasal cavity. Swelling of the lymphatic tissues in the pharynx, of the œsophageal and intestinal follicles, cervical lymphatic glands, and of the lymph glands in other regions of the body (the lymph glands are marrow-like, grayish white, and well circumscribed).

General anemia, acute splenic tumor, calcified tuberculous subpleural focus in the right lower lobe with adhesion to the costal pleura; œdema of the brain and of the lungs. Multiple punctiform hemorrhages in the mucous and serous membranes, as well as in the lymphatic tissues. Moderate arterio-sclerosis and atheroma of the aorta. Height, 175 cm.; brain weighs 1300 grams. The thymus is replaced by a large fatty mass. Patulous foramen ovale. Adipositas. Adenomas in the thyroid gland, to the right and above the middle lobe and not separated from it. Bone marrow (of femur) $\frac{3}{4}$ red. Grayish-yellow coagula in the bloodvessels. Chronic internal hydrocephalus with granulations on the ependyma. The body was muscular and apparently well nourished.

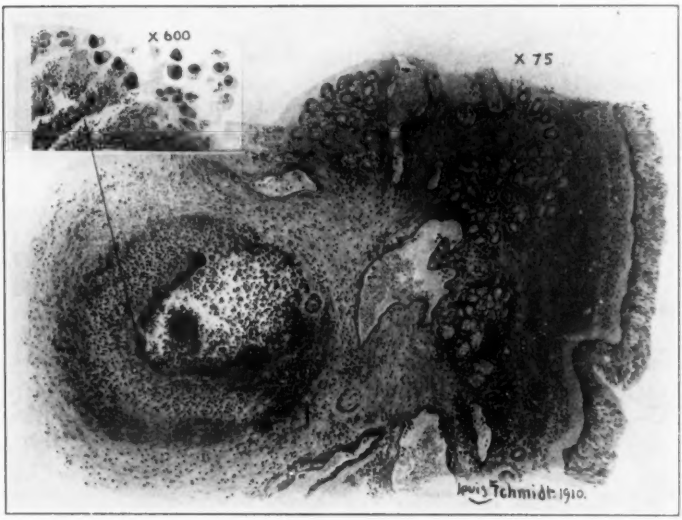
Upon opening the skull by the Ghon method a hemorrhage was seen in a very large sphenoidal sinus which measured 4.5 x 2.75 cm. Both the anterior and posterior walls of this sinus were extremely thin. On account of the numerous punctate hemorrhages over the entire nasal mucous membrane, more especially that covering the turbinates, I removed fragments from the middle and inferior turbinates for microscopic examination. Dr. Bartel also removed a number of fragments of tissues from almost every part of the body and subsequently confirmed the results of our examinations.

Microscopic Appearance. The affected mucous membrane is swollen to four or five times the ordinary thickness. The surface, in the sections examined, shows a decided desquamation and proliferation of the epithelium, which presents in places considerable irregular thickening and projection, while elsewhere the layer is thin, with loss of superficial cells. In the drawing (Fig. 1), proceeding from the right-hand side, there is, first, a layer of stratified columnar epithelium, beneath which a thin layer of œdematous tissue is succeeded by a superficial layer of round-cell infiltration. Adjacent to this there are a number of mucous glands and large injected vessels. To the left there is an abscess of the submucosa, with a loose, purulent centre; a border zone of the wall, showing in horseshoe form, largely made up of streptococci, to the outside of which is the inflammatory reaction zone of the wall.

In the section from which this drawing was made there were four of these abscesses present. It is a conservative estimate, therefore, to assume that there were at least one hundred similar abscesses in this man's nares.

Particularly in the thickened portion the epithelium is somewhat infiltrated with polymorphonuclear leukocytes. The tissue beneath the epithelium, down to the bone, is the seat of marked injection of the vessels, of focal hemorrhages, of a moderate diffuse œdema, together with a considerable mononuclear collection, especially near the surface, and a less marked diffuse polynuclear leukocitic infiltration. In the limits of the section four or five small abscess cavities occur, in the inner portion of the wall of each of

which are large masses of Gram positive streptococci. Scattered through the tissue small foci of similar organisms are occasionally met. Toward the surface of the membrane there are numerous phagocytic cells containing blood pigment granules, and in the looser portions of the structure, especially about the abscess, a fine fibrinous reticulum occurs. The mucous glands are of the usual distribution and number, frequently showing mucin in the interior.



Section through the mucosa and submucosa of a turbinate bone from a case of lymphatic leukemia showing hyperemia and inflammatory infiltration and a small suppurative focus, on the inner margin of the wall of which masses of streptococci are seen. These are shown at higher magnification in the upper left-hand corner, the reference line indicating the position in the larger field.

In the preparation examined it has been impossible to recognize, in the blood of the hemorrhagic foci or in that within the blood-vessels, features characteristic of leukemia. An especial round-cell infiltration, extending through the superficial portion, is probably rather inflammatory than of a lymphatic leukemic formation. The diagnosis of lymphatic leukemia in this case was made very positively from an examination of tissues elsewhere in the body. In another case, however, sections of the inferior turbinate showed undoubted evidence of a leukemic condition of the blood.

The history of a second case was as follows:

J. L., a coachman, aged twenty-eight years; died of pneumonia and leukemia. The mucous membrane of the turbinates was very much congested. I removed the posterior end of the left inferior

turbinate, sections of which, under the microscope, gave a clear picture of chronic inflammation. The bloodvessels were considerably enlarged and the lumen of the corpora cavernosa filled with leukemic blood.

Dr. James B. Herrick,[1] of Chicago, has reported a case of multiple abscesses of the brain in a case of leukemia. The patient was a young man, aged twenty-three years, who came under observation in March, 1905, and, despite the fact that the diagnosis was made promptly, he died in November of the same year. The abscesses were about the size of a ten cent piece in this case and were scattered everywhere throughout the cerebrum.

That interstitial hemorrhages are the rule rather than the exception in leukemia is a well-known fact, but that these hemorrhages, no matter how minute, may rapidly develop into abscesses is of considerable practical importance.

[1] Multiple Hirnhämorrhagien bei Leukämie, Festschrift, Dr. Hans Chiari, Wien, 1908, 317 to 333.

REVIEWS.

PREPARATORY AND AFTER-TREATMENT IN OPERATIVE CASES. By HERMAN A. HAUBOLD, M.D., Clinical Professor in Surgery in the New York University. Pp. 650; 429 illustrations. New York: D. Appleton & Co., 1910.

THE preface of this work sets forth convincingly the reason for its preparation and, unlike most prefaces, is not only logical, but readable. It emphasizes the fact—which is familiar to all surgeons—that the general practitioner is, as a rule, unfitted to make the arrangements for an important operation and to take care of the wound, and sometimes of the patient, during the critical stage of the after-treatment. That this is often not in the least the fault of the medical man, but is due to unavoidable lack of early technical training, or, even after such training, to the exacting demands of general practice, does not alter the situation. The result is that not only during, but often both before and after an operation, the regular medical attendant is to an extent displaced and supplanted, sometimes quite unintentionally and unavoidably belittled, not by the surgeon, but by his assistants and his special nurses, whose work during the preliminary and the after-treatment seems, by implication, to render the services of the medical man unnecessary or valueless. Partially as an outcome from this situation, and partially from motives that are as obvious as they are unworthy, has arisen the widespread and essentially dishonorable practice of giving the medical attendant a percentage of the surgical fee, a custom even more discreditable to a learned profession than is the "contingent fee" of our legal brethren.

The author says truthfully: "Much of this is due to the fact that neither the practitioner nor the patient have been properly educated in the matter. The practitioner has not devoted much energy to keeping in touch with modern methods of preparing patients for operation, and the patient has not been taught to understand that the work connected with or the services necessary in this regard is special work and demands a special fee." He adds that it is even more true of the after-treatment of operative cases. "The surgeon does his operation, takes the fee, and the practitioner is compelled to carry out the after-treatment of the case at the usual rate of charge for a visit."

It is a pity that so well written a preface should be marred by the next sentence in which the arrangement is said not to be "equable"—for "equitable"—and to stand in a causative relationship to the methods "of handling the financial end of the surgical proposition." an example, in spite of the undoubted truth of the assertion, of the use, or misuse, of several words that undoubtedly belong in the "Inferno" of our contemporary, "The Bookman."

The author continues, modestly, to say that if the book achieves nothing beyond placing the relationship of the practitioner and the surgeon to each other and of both toward the patient on a better basis, he will feel that a worthy object has been attained.

He has, however, achieved much more, and has written a book likely to be of great use not only to the genearl practitioner who is occasionally brought into close relation with a surgical case, but also to the young surgeon whose ideas require systematization, and even to the experienced surgeon about to take charge of an unusual case, or of an ordinary case under unusual circumstances.

Portions of the book particularly to be commended are those devoted to general considerations in non-urgent cases and to the preparation of the room, the bed, and the patient. The subject of diet in relation to operation is fully and adequately considered, although the author possibly over-emphasizes his objections to the "starvation" of patients a day or two before operation. If the expression is taken literally there is, however, no cause for cavil. The principles and details of feeding after operation are well summarized. The technique advised throughout the book is thorough, sensible, and dependable. Each surgeon is apt to have preferences in this respect, and, as might be expected, there is room for honest differences of opinion; but the methods here described, if intelligently employed, should, practically without exception, give satisfactory results.

The treatment of shock and of secondary hemorrhage, the dressing of the operative wound, the treatment of special symptoms, as thirst and pain, are thoroughly dealt with, and the special methods indicated in the preparation and in the after-treatment of particular operations, are fully and often minutely described. There is a useful chapter on artificial limbs. Another on anesthesia, general and local, would have perhaps added to the value of this excellent book, as, especially in emergencies, the medical attendant is so often the anesthetist. The illustrations are well chosen and well made. The book admirably covers its chosen ground and should be welcomed and widely read by the profession at large. J. W. W.

THE PRESCRIBING OF SPECTACLES. By ARCHIBALD STANLEY PERCIVAL, M.A., M.B., B.C., Cantab., Senior Surgeon to the Northumberland and Durham Eye Infirmary. Pp. 159; 24 diagrams. Bristol: John Wright & Sons, Ltd.; London: Simpkin, Marshall, Hamilton, Kent & Co., Ltd.; New York: William Wood & Co., 1910.

THIS book must have been written for those who are already acquainted with the subject; it is far too concise for the beginner, so that all it is likely to impart to the reader capable of understanding it are certain views and practices peculiar to the author, or perhaps to his country generally; thus, in the shadow test the concave mirror is preferred to the plane; two or three drops of a solution containing 8 grains of homatropine and 4 grains of cocaine to the ounce every quarter of an hour will paralyze the accommodation. In ordering glasses for hypermetropia, it is usually sufficient to deduct I D from the total hypermetropia if atropine, and 0.75 D if homatropine is used. The author would do away with the word presbyopia, under which term, if used at all, should be included hyperopia acquisita. The two-thirds rule is recommended in the correction of defective accommodation. He objects to Landolt's allowance of one-third of the absolute range of convergence.

The last chapter in the book gives the optical formulæ as applied to the constants of the eye, correcting lenses, size of images, etc., very interesting to the reader who is fairly well acquainted with elementary optics and understands the meaning of the formulæ and how they are derived; to others, among whom we fear is to be placed the great majority of students of ophthalmology, it is a closed chapter. Several tables are added in an appendix; one gives the squares of numbers from 10 to 100, another the values of the trigonometrical functions. T. B. S.

A TREATISE ON DISEASES OF THE EYE. By JOHN ELMER WEEKS, M.D., Professor of Ophthalmology in the University and Bellevue Hospital Medical College (Medical Department of the New York University). Pp. 944; 528 engravings and 25 full-page plates in colors. New York and Philadelphia: Lea & Febiger, 1910.

THIS extensive work covers the entire field of ophthalmology. The style is singularly clear. Those parts which are usually difficult of comprehension, such as the development of the eye and certain parts of the anatomy, are so presented that even the beginner should have no difficulty in getting clear ideas. It is, moreover, thoroughly up to date. The microbic affections, as is to be expected

from its distinguished author, are especially well presented. Even the trachoma bodies are figured and described. We have failed to find any reference to the Wassermann reaction.

The removal of the clear lens in high myopia is stated to be now fully recognized in suitable cases, although it has not fulfilled the expectations of those who closely followed Fukala. While we are told that it should be employed only in carefully selected cases, how the selection is to be made is not clearly stated. The author advises, as a rule, that the total hyperopia should not be corrected except in cases of marked esophoria and strabismus. Early full correction of myopia is advocated. Before prescribing prisms for the correction of heterophoria, the advice is given to note the behavior of the eye under the correction of the ametropia, as the heterophoria, especially esophoria, may disappear when the error of refraction has been corrected. The necessity for a cycloplegic may be inferred from the text; it might be as well to make a direct statement to that effect. The most practical test for determining the refraction in intelligent patients is declared to be subjective examination with the trial lenses; the objective tests are valuable aids.

Among the newer subjects dealt with is arteriosclerosis. A short chapter is devoted to disease of the accessory sinuses as related to the eye.

The work is singularly free from slips of the pen and careless proofreading. Indeed, we have not come across a single instance of the latter. We do note, however, an inversion of words on page 122, where it is stated that in case of paralysis of the third nerve the *afferent* part of the reflex arc is interfered with, while the *efferent* part is intact.

The mechanical execution of the book leaves nothing to be desired. The type and paper are admirable. The numerous engravings are just what is needed to complete the descriptions. Most of the colored plates, of which there are 25, are unexceptional.

The work is to be recommended to everyone interested in the subject, from the undergraduate in medicine to the specialist in ophthalmology, for both of which classes the author tells us he has designed his work. T. B. S.

THE PREVENTION AND TREATMENT OF ABORTION. By FREDERICK J. TAUSSIG, A.B., M.D., Lecturer in Gynecology in the Washington University; Obstetrician to the St. Louis Maternity Hospital, etc. Pp. 172; 59 illustrations. St. Louis: C. V. Mosby Co., 1910.

THERE can be no difference of opinion of the importance of the subject of which this little volume treats, since there is probably

no one condition more often met with by the general practitioner, few that give more concern to the conscientious man, and none which is treated more casually by a large number of the profession. The truth of all these statements will, we believe, be vouched for by all those doing work in the diseases of women. Certainly the casual attitude of the general profession has been painfully evident to all who do much consultation work along these lines. We therefore welcome any and all attempts to impress upon the profession their responsibilities in this direction. The author of this book claims no originality for anything except the manner of presentation. His experience has eminently fitted him for the preparation of the volume, and he has produced a very readable book, our only criticism being that the case histories might have been omitted without lessening the value of the work.

The book is well written and the deductions with regard to treatment are for the most part entirely sound, though we would have omitted the use of gauze packing as a means of induction of labor. We believe that unless a man is competent to use instruments he had better not perform the operation at all, because of the added danger of infection which the method under discussion undoubtedly causes, especially in the hands of the occasional operator. This restriction is, however, hardly fair, as there is good authority in favor as well as opposed to this method. The whole subject is admirably covered, and besides the chapters upon diagnosis, symptomatology, etiology, and pathology, the inclusion of which are, of course, predicted by the title, chapters have been included upon prophylaxis before and during pregnancy, operative technique, sepsis, and perforation. In addition, missed abortion, mole pregnancy, and therapeutic abortion, as well as ergot and its preparations, are discussed in an appendix of fifteen pages. W. R. N.

SPONDYLOTHERAPY. SPINAL CONCUSSION AND THE APPLICATION OF OTHER METHODS TO THE SPINE IN THE TREATMENT OF DISEASE. By ALBERT ABRAMS, A.M., M.D., Consulting Physician to the Mount Zion and French Hospitals, San Francisco. 97 illustrations. San Francisco: The Philopolis Press, 1910.

THIS is rather a curious book which has not altogether repaid the time spent in its perusal. The following quotation from the preface may serve as an indication of the mental attitude in which the preparation of the book was approached. "The author was lead to a deeper study of spinal therapeutics in investigating various visceral reflexes which bear his name;" and further: "The successful

practice of spondylotherapy requires knowledge, observation, and experience of the highest kind." Turning to the end of the book, a bibliography of sixty-seven numbers is given, of which twenty-two are references to articles or books by the author. The subject appears to have some affinity with osteopathy and with the methods of one or two other schools. There are numerous tables selected, as a rule, from other authors, and some methods that appear to be distinctly original, particularly spondylography. Some of the statements appear to one not well versed on this subject rather sweeping. For instance, the painful centres, for the finding of which an ingenious method is given, are said to be so pathognomonic of certain ailments that the physician can almost make a diagnosis from the reflex centres involved. Another is that when one finds the spinal tenderness associated with some organic disease of an organ, as, for example, a gouty toe, or an ulcer of the stomach, if the area of spinal tenderness be frozen, the pain will disappear in the affected organ. If the freezing be repeated, apparently the ulcer or gout will get well, which is explained as follows: "Now, to the average physician it would be ridiculous that freezing over the area of tenderness was anything more than a palliative measure, yet sober thought endows analgesia with curative action." The reviewer hesitates to express his own thoughts upon this subject. This method is also effective, apparently, for persistent coughs.

Respiratory ataxia, a condition which has not received the attention the author believes it deserves, and which is capable of producing a remarkable group of symptoms, is thoroughly described. There is a very curious classification of the different types of backaches, for one of which, at least, he accepts the uric acid etiology. He is an enthusiastic believer in the sinusoidal current. He holds that lumbar puncture will relieve headache due to an augmented intercranial pressure.

There is a description of a variety of pseudovisceral diseases which yield promptly to the freezing method. There is also a long chapter upon the forms of cardiovascular disease that can be relieved or cured by spinal treatment, including particularly cardiac insufficiency, the changes in tension of the blood vessels, and even, apparently, aneurysm of the aorta. There are also sections on the respiratory and gastro-intestinal systems. The spinal methods of treatment are available in gastroptosis, intestinal intoxication, constipation, and a variety of other conditions.

The author is apparently an enthusiast, but it is probable that a considerable portion of the medical profession will await more conclusive demonstration than the perusal of his book before adopting his methods generally. At any rate, the reviewer has the feeling that the elaborate series of visceral reflexes described has hitherto strangely escaped common medical knowledge. J. S.

GRUNDRISS UND ATLAS DER SPECIELLEN CHIRURGIE. By PROFESSOR DR. GEORGE SULTAN. Vol. II, pp. 624; 261 illustrations and 40 colored plates. Lehmann's Medical Hand-Atlases, Vol. xxxvii. Munich: J. F. Lehmann, 1910.

THE author apologizes for the long time (three years) elapsed since the appearance of the first volume of this popular work; but this delay has permitted him to include accounts of quite modern surgical methods only now coming into general use, such as arterial suture. Yet certain procedures are advised which many surgeons no longer regard as of value. Among such may be mentioned separate suture of the mucosa in intestinal anastomosis; irrigation in peritonitis; and the use of Bardenheuer's weight extension for fractures of the surgical neck and shaft of the humerus, the patient being confined to bed. There is no mention of Ruggi's inguinal method for the radical cure of femoral hernia; nothing is said of arthroplasty; and the section on chronic non-tuberculous arthritis is devoid of what little advances in classification and diagnosis have been secured by American surgeons. A bad prognosis is given for elbow fractures. In accordance with the plan of these Hand-Atlases, there is no attempt made to include pathology or etiology except as they bear directly on diagnosis; chief attention being given to symptoms and treatment. The first volume covered the head, thorax, and spine; while the present volume, larger by nearly 200 pages, includes the abdomen, male genito-urinary organs, and the extremities. The numerous illustrations are well chosen, and the colored plates unusually good. A. P. C. A.

CONGENITAL DISLOCATION OF THE HIP. By J. JACKSON CLARK, M.B. Lond., F.R.C.S., Senior Surgeon to the Hampstead and North-West London Hospital. Pp. 92; 55 illustrations. London: Baillière, Tindall & Cox, 1910.

THIS is largely a personal record of the experience derived by the author from the manipulative treatment of forty cases of congenital dislocation of the hip. He gives entire credit for the development of the "bloodless" method to Lorenz (1895), damning with faint praise the pioneer work of Paci, in the following words: "There is no doubt, however, that the work of Paci had much influence in bringing about this great advance in surgery." Clarke, like many other surgeons, seems totally unaware that Lorenz as well as Hoffa was an early advocate of the "bloody" method, and that Paci "was publishing his work by the bloodless method from 1888 to 1893." "In 1894, at the International Congress in Rome, Lorenz and Hoffa came prepared to fight for their respective cutting operations,

but Paci by this time had systematized his work. He gave his results on 23 cases of congenital luxations, besides 5 of pathological and old traumatic ones; also those of ten other surgeons who had used his method. He submitted a specimen showing a perfect antemortem reposition, and wound up by performing his bloodless method on a patient before the assembled surgeons. The effect might almost be described as ludicrous. No more was heard of cutting operations. Lorenz, in the following year, modified Paci's procedure, and travelled far and wide exploiting it." (G. G. Davis.)

Among Clarke's 40 cases, he considers the results established in the first thirty patients. "These presented 39 dislocations, of which 18 were double and 21 were single; of the 18 double ones, 12 are cured and 2 are anteverted in a stable position, and 4 failed. Of the 21 single joints, 17 are cured, 1 is anteverted, and 3 have relapsed; the total being 29 out of 39 cured, 3 stable anteversions, and 7 failed." For cases which relapse, he has devised an operation consisting in incising the capsule posteriorly, elevating the periosteum and cotyloid ligament from the upper and posterior margins of the acetabulum, and pleating the capsule to these raised margins after reduction of the femoral head. This method he has employed successfully in 2 cases. For irreducible luxations he knows of no satisfactory treatment; beyond the slight relief to be secured from orthopedic apparatus, he regards these cases as hopeless. He seems to be unaware of the advances in the treatment of such cases inaugurated by G. G. Davis, Ludloff, and others. A. P. C. A.

THE PSYCHOLOGY OF DEMENTIA PRÆCOX. By Dr. C. G. JUNG, Privat-Docent in Psychiatry in the University of Zurich. Authorized Translation, with an Introduction by FREDERICK PETERSON, M.D., Professor of Psychiatry in Columbia University, New York, and A. A. BRILL, Ph.D., M.D., Assistant in Psychiatry in Columbia University, New York. Pp. 153. New York: The Journal of Nervous and Mental Disease Publishing Company, 1909.

THIS is the third of the Monograph Series produced by the *Journal of Nervous and Mental Disease*. Its translators, Frederick Peterson, M.D,. and A. A. Brill, M.D., while giving in their preface credit to Kräpelin for having introduced new life into psychiatry, insist that he only offered a general and superficial view of the subject and that he did not enter at all into psychology. The present work is the result of three years' experimental labor and clinical observation, and is along the same lines as Freud's well-known work on hysteria. In fact, the author admits that his attention was

drawn to the subject by the stimulation received from Freud's work. The subject matter is divided into five chapters and a conclusion. In the latter the author frankly admits that this work is simply a beginning, and is only an exposition of his own method of diagnostic association study in mental disease, and especially in dementia præcox. The last chapter is of special value, inasmuch as the author gives in detail his method of examining a case and the deductions at which he arrives. The work is well done, and should be read by everyone interested in mental diseases. T. H. W.

De l'Usage en Clinique de l'Ultra-microscope en particulier pour la Recherche et l'Etude des Spirochetes. Par le Dr. J. Comandon, de la Faculté de Médecine de Paris licencié des Sciences. Paris: Steinheil, 1909.

To one who is using the dark field illumination for routine or special work this monograph will probably be of some service. It does not, however, bring to light many new facts of great interest and, therefore, must have a limited usefulness. The apparatus for dark field illumination is first described in sufficient detail, and then follows a number of chapters upon the use of this apparatus in the search for spirochetes in syphilis and the value of the method in the study of other forms of spirillosis. Certain observations upon the form and behavior of spirochetes in various fluids are reported, and the pamphlet ends with a study by the dark field illumination of a number of body fluids. W. T. L.

The Open-air or Sanatorium Treatment of Pulmonary Tuberculosis. By F. Rufenacht Walters, M.D., B.S., M.R.C.P., F.R.C.S., Physician to the Crooksbury Sanatorium, etc. Pp. 323; 29 illustrations and tables. New York: William Wood & Co., 1909.

Dr. Walters prepared the book with a twofold object—that the first part be concerned with matters which an intelligent patient should know, and that the second part be concerned with the technical side for the assistance of the physician. Numerical references are made in many places in Part I to the more technical Part II. In the former there are several illustrations which show the proper methods of building floors, verandas, windows, shutters, screens,

etc., in sanatoria and improvised out-door shelters for consumptives. The subject of dietetics receives special attention by the presentation of about two dozen "useful recipes." Aside from the usual exposition of the technical side of tuberculosis, Part II contains a rather exhaustive table dealing with food values and their importance in tuberculosis. To the patient the subject in Part I is stated in a simple and thoroughly comprehensible manner; moreover, the illustrations lend attraction. However, one must take issue with certain statements relative to disinfection. It is well known that carbolic acid and bichloride of mercury are ineffectual in the disinfection of furniture and sputum respectively. To the physician the subject in Part II is presented systematically and comprehensively, though very briefly. On the whole, the book is meritorious, but one questions the value of adding Part II to a volume evidently prepared primarily for the guidance of the consumptive patient.

W. T. C.

REFRACTION AND MOTILITY OF THE EYE, WITH CHAPTERS ON COLOR BLINDNESS AND THE FIELD OF VISION, DESIGNED FOR STUDENTS AND PRACTITIONERS. By ELLICE M. ALGER, M.D., Adjunct Professor of Ophthalmology in the New York Postgraduate Medical School; Ophthalmologist to the New York Dispensary, etc. With 122 illustrations. Philadelphia: F. A. Davis Company, Publishers, 1910.

ABOUT two-thirds of this work is devoted to refraction and one-third to motility; the latter is the better. The author properly insists that the postgraduate student should master the principles (of optics and refraction) before entering a school; it is questionable whether the preliminary chapter upon optics is clear or full enough to be comprehended by self-study by one who knows nothing of the subject. As an acquaintance with the principles of optics is at the foundation of all further progress in understanding normal and abnormal refraction and accommodation, it is desirable to make this chapter as clear as the subject will possibly permit, even at the expense of many pages, illustrations of all kinds, and repetition if necessary.

Where extreme and contradictory views prevail the author wisely chooses a middle course. We could wish he were rather more emphatic upon the necessity of cycloplegics. The "two-thirds" rule in presbyopia, which is not mentioned at all, is surely superior to "ordering a convex glass strong enough to bring the near point to ten inches." We should like to see a reason for the author's preference for placing the light above the patient's head in the shadow test, rather than close to the mirror, which latter alone permits

practical coincidence of the point of reversal with the virtual source of light—an important detail which makes so strongly for accuracy. In directions for ordering glasses in hypermetropia we find no reference to allowance for range, but a series of rules, most of which we believe would fall away if the necessity of such an allowance were generally understood. T. B. S.

SURGICAL ANATOMY. By JOHN A. C. MACEWEN, B. Sc., M.B., C.M., Senior Assistant to the Regius Professor of Surgery in the Glasgow University. Pp. 478; 61 illustrations. New York: William Wood & Co., 1910.

THIS manual of surgical anatomy is designed for the instruction of students of surgery and anatomy, and covers the matter usually embraced in a course on the subject. The volume, which is of quite modest size, is divided into five sections, discussing successively: (1) Head and Neck, Vertebral Column, Brain and Spinal Cord. (2) Thorax. (3) Abdomen and Pelvis. (4) Lower Extremity. (5) Upper Extremity. The illustrations are few in number, but well selected, and serve well by their diagrammatic, if not always artistic, clearness to inculcate the salient points accentuated in the text.

It is perhaps hypocritical to wish that an author who writes upon surgical anatomy should have written instead upon applied anatomy; but the student and practitioner so often look for the practical application of anatomical facts in text-books on surgical anatomy, and fail to find just what they want, that we wish the author of the present volume had paid more attention to the applied aspects of his subject, and less to regional anatomy. This is not meant to imply that there are no practical applications, for there are numerous such, and very excellent too; but he has not, we venture to suggest, a just appreciation of the distinction between applied and merely surgical anatomy. There are many excellent text-books and manuals upon the latter subject, but a work of the highest type on applied anatomy has yet to be published.

There are a few typographical errors (*e. g.*, *visci* for *viscera*, p. 148; *errigentes* for *erigentes*, p. 183; *eighth* for *eighteenth*, p. 402, line 3) which do doubt will be corrected in another edition; and the use of the term *incontinence* (p. 148) of urine and feces, when in reality *overflow* is intended, will perpetuate confusion in students' minds as to the effects of lesions of the spinal cord above and below the lumbar enlargement, in which are the centres for the bladder and rectum. The distinction to be drawn is that in damage to the cord *above* these centres there is overflow of urine and feces, as the voluntary impulses cannot reach the spinal centres, with the result that

the centres act automatically (a common phenomenon); whereas if the centres themselves, or the nerves between them and the bladder or rectum are damaged, then there is true *incontinence* (very rare).

A. P. C. A.

MAMMALIAN ANATOMY, WITH SPECIAL REFERENCE TO THE CAT. By ALVIN DAVISON, Ph.D., Professor of Biology in Lafayette College. Second edition. Pp. 246; 114 illustrations. Philadelphia: P. Blakiston's Son & Co., 1910.

THE author states that "when the first edition of this book was published, less than a half dozen medical schools in the United States required the entering student to have any knowledge of the structure of the lower animals. To-day a knowledge of mammalian anatomy or zoölogy is considered one of the first requisites for entering upon an intelligent study of medicine." To meet such requirements the present volume appears to be well adapted; and it may also be found useful, as the author suggests, to the student of psychology and teachers of elementary physiology in high schools. In the present edition the BNA nomenclature has been cautiously introduced; and new figures and a glossary have been added.

A. P. C. A.

L'HYPOPHYSE ET LA MÉDICATION HYPOPHYSAIRE. By ARTHUR DELILLE. Pp. 336. Paris: G. Steinheil, 1909.

THIS monograph considers the functions and pathology of the pituitary as determined by a series of experimental and clinical studies, executed under the direction of L. Renon, and extending over a period of three years. The first part of the book takes up the anatomy, histology, physiology, and chemistry of the hypophysis and its secretion; the second part is devoted to experimental researches, in which the action of extracts of pituitary is considered in relation to various physiological processes; the third part deals with clinical observations and observations of the effects of the administration of extracts of pituitary in different diseased conditions; and the fourth part discusses the indications for medication with pituitary extracts.

This contribution to a subject but little understood represents much careful work, especially along experimental lines, and the subject matter is presented in a concise and conservative manner. Some of the most important conclusions of the author are as follows:

Histology demonstrates that the anterior lobe of the pituitary body possesses decided glandular activity, and the posterior lobe is devoid of such activity. On the contrary, experiments prove that the extract of the posterior part is physiologically active, and that of the anterior portion gives no apparent results. The extract of the posterior lobe (or of the entire gland) causes elevation in arterial tension and vasoconstriction, less accentuated than that from the use of suprarenal extract, but of longer duration. On the kidneys, the effect is at first vasoconstriction, but later vasodilatation and diuresis. Physical development and intelligence are stimulated, thyroid activity is diminished, and suprarenal function is increased. Clinical observations practically coincide with experimental results and pituitary administration is suggested in conditions presenting low arterial pressure, tachycardia, sensations of heat, profuse perspiration, oliguria, anorexia, asthenia, insomnia, arrested development, etc. It is also recommended that the daily dose should not exceed one-half of a fresh gland of the beef or its equivalent.

S. D. I.

A Text-book of Pathology. By Joseph McFarland, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College, Philadelphia. Second edition (pp. 956); 437 illustrations. Philadelphia and London: W. B. Saunders Co., 1910.

A Text-book upon the Pathogenic Bacteria. By Joseph McFarland, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College, Philadelphia. Sixth edition (pp. 709); 211 illustrations. Philadelphia and London: W. B. Saunders Co., 1910.

Dr. McFarland's two books—the one on *Pathology*, the other on the *Pathogenic Bacteria*—have become standards in their respective fields, so that it becomes necessary only to discharge the pleasant duty of recording the publication of new editions. Both books have been considerably revised; the results of recent studies and investigations and the real additions to our knowledge in pathology and bacteriology have been incorporated. Both, therefore, may be highly commended as representative of present-day knowledge of the subjects of which they treat. Perhaps some day Dr. McFarland will add to his book on the *Pathogenic Bacteria* a section on the pathogenic zoöparasites, the pathogenic as well as economic importance of which is becoming almost daily increasingly manifest.

A. K.

PROGRESS

OF

MEDICAL SCIENCE.

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UNDER THE CHARGE OF

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A New Sign of Inflammation of the Meninges.—SIGNORELLI (*Rivista Critica di Clinica Medica*, 1910, No. 15) describes the so-called retro-mandibular tender point as a constant sign of meningitis. This point is located behind the superior extremity of the inferior maxilla, below the lobule of the ear, and in front of the mastoid process. In healthy individuals this point is sensitive, but in cases of meningitis pressure with the index finger elicits extreme pain and provokes contractions of the facial muscles: it is apparently the trunk of the facial nerve which is sensitive. The sign is present throughout the attack. It often precedes the stiffness of the neck and the Kernig sign. In tuberculous meningitis the muscular contractions, after pressure at this point, occur even when the patient is paralyzed and comatose.

Chronic Antimony Poisoning in Typesetters.—SCHRUMPF and ZABEL (*Archiv f. exp. Path. u. Pharm.*, 1910, lxiii, 242) have had the opportunity of observing a large number of typesetters during the last year or more, and were struck with the variety of lead poisoning among them. Many of the typesetters, however, exhibited a clinical picture characterized by a tired expression of the face, complaints of nervousness, irritability, insomnia, exhaustion especially in the morning, vertigo, headache, general and local muscular pains, neuralgic pains in the extremities, nausea and a tendency to vomit, with disturbance of the stomach and bowels, constipation. Varying degrees of intensity were observed. The majority of those affected were young workmen between the ages of fifteen and thirty-five years, who had been employed as typesetters for periods of one-half to thirty-one years. Schrumpf and Zabel were at first inclined to regard their symptoms as manifestations of

plumbism. They found, contrary to the rule in lead poisoning, that the blood pressure was not elevated, that the blood contained no basophilic granules, nor was there leukocytosis, and that no albumin or bile pigment could be detected in the urine. In fact, the blood pressure, they say, was rather low, there was leukopenia with a marked eosinophilia (10 to 25 per cent.), there were no basophilic granules in the red cells, though slight polychromatophilia was seen, and in one case a few normoblasts, and the urine was normal. In typesetters without any complaints, and in whom physical examination revealed no organic disease, the blood was normal, except for moderate increase in the eosinophilic cells (up to 9 per cent.). They concluded, therefore, that a diagnosis of lead poisoning was not justified. On inquiry, the authors found that the mixture used in covering the type contained lead, 70 to 80 per cent., tin, 5 per cent., and antimony, 15 to 20 per cent. As tin is of no importance toxicologically, their attention was directed to the antimony. Experiments on rabbits showed that antimony causes a rather marked reduction in the number of leukocytes, with an eosinophilia up to 25 per cent; no other changes were found in the blood. (Arsenic, which is an impurity often present in antimony, caused some leukopenia, but the effect was less marked than with antimony.) With Marsh's test, an antimony mirror was obtained from the feces of the poisoned rabbits. Schrumpf and Zabel then examined the stools of their patients, and in two of them they obtained an antimony mirror, removing practically all doubt as to the nature of the intoxication. The prognosis of the condition is good. Absence from work for two or three weeks, with a milk diet and exercise, sufficed to relieve the symptoms in the majority of instances. After leaving their employment there was an increase in the number of leukocytes in the patients' blood.

The Treatment of Malaria with "606."—WERNER (*Deut. med. Woch.*, 1910, xxxvi, 1792) has used "606" in 22 cases of malaria (11 each of tertian and estivo-autumnal fever) which were resistant to quinine. With 0.6 to 0.7 gram of "606" the tertian fevers are apparently cured. The estivo-autumnal infections proved resistant to the remedy; the asexual forms disappeared temporarily from the blood, but reappeared in about six days in one-half of the cases. The drug acts in about twenty-four hours. Werner recommends "606" in those cases in which the parasite is resistant to quinine and also in patients who are intolerant of quinine. He recommends that the dose, 0.6 gram, be given in part subcutaneously and the remainder intravenously.

A New Method of Administering "606."—KROMAYER (*Berl. klin. Woch.*, 1910, xlvii, 1698) has devised a new method of administering "606" which has some obvious advantages. He takes 3 grams of "606" and rubs it in a mortar with liquid paraffin until the drug is in a finely divided state. It is then transferred to a glass-stoppered bottle of 50 c.c. capacity, and liquid paraffin is added to make the volume 30 c.c., so that 1 c.c. equals 0.1 gram of "606." It should be kept in the dark. Before using, the suspension must be shaken until no sediment remains at the bottom of the bottle. The needles for injection should be of wider bore than those usually employed in giving mercury salicylate, and are best kept in a Petri dish in fluid paraffin. They must, of course, be sterilized

each time before using. The injection must be made very slowly to avoid tearing the tissues. Kromayer has used this method in 100 cases without a single instance of pain or local swelling. Ambulant cases may receive the injection unless there are definite contraindications. Clinical results indicate that the arsenic is rapidly absorbed and urinary examination confirms this view.

The Treatment of Strychnine Poisoning by Insufflation and Curare.—SHAKLEE and MELTZER (*Berl. klin. Woch.*, 1910, xlvii, 1776) find that 0.4 mg. strychnine per kilogram of body weight is invariably fatal to dogs, death being due to asphyxia from spasm of the respiratory muscles. They have employed insufflation and curare in the treatment of experimental strychnine poisoning in dogs. The animals were given 0.5 to 1 mg. per kilogram; 0.9 to 1 mg. always proved fatal. With doses of 0.8 mg., twice the lethal dose, recovery of the animals was obtained in 13 of 22 instances. The only visible sign of life in the animal was the heart beat. The dose of curare employed was 1.5 mg. per kilo. Infusion of Ringer's solution hastened the elimination of both strychnine and curare. Shaklee and Meltzer suggest this treatment in suitable cases of poisoning in man. The surviving dogs were sacrificed at the end of a few weeks or months, and showed no ill effects from the insufflation in the trachea, bronchi, or lungs, though the experiments lasted three and one-half to seven hours.

The Effect of Malignant Tumors upon the Blood.—BARADULIN (*Folia Hæmatol.*, 1910, ix, 407) reports a careful and detailed study of the blood of 72 patients suffering with cancers in various parts of the body, and of 16 patients with sarcomas. He has summarized the findings in the carcinomatous patients, grouping the cases according to the location of the newgrowth in the body. In the general summary of all cases of carcinoma the following facts are emphasized (the individual summaries possess greater value but would require too much space): (1) Decrease in the hemoglobin is proportionate to the cachexia; in many cases it sinks to an extremely low point. The decline in hemoglobin is also dependent largely on the location of the cancer. The size of the tumor is without influence and its duration is also of little moment. Tumors which do not interfere with the respiration or digestion may grow for months with little or no reduction in hemoglobin. (2) The number of erythrocytes is decreased, but to a less extent than the hemoglobin, so that (3) the color index is below 1. (4) In the later stages of cancer of the internal organs, microcytes, macrocytes, and poikilocytes appear. They are the more numerous, the severer the cachexia. Nucleated red cells with polychromasia may be seen. (5) With slowly growing neoplasms, leukocytosis is slight or is missed altogether. On the contrary, rapidly growing tumors cause leukocytosis, with increase of the polymorphonuclears and lymphocytes in the earlier stages, later in the disease, of the mononuclears. Those tumors which ulcerate, become infected, and lead to hemorrhage, present leukocytosis with increase of the mononuclears. (6) The specific gravity of the blood is lowered in proportion to the diminution in hemoglobin and red count. (7) The alkalinity is reduced with rapidly growing tumors. With sarcomas there is moderate anemia with low color index. Leukocytosis

was observed in all cases. Sarcoma of the soft parts occasions an increase of the polymorphonuclears at the cost of the lymphocytes. In lymphosarcoma, the condition is just reversed. Bone sarcoma is accompanied by increase of transitionals and eosinophiles, with decrease of polymorphonuclears. Myelocytes are found. The specific gravity and alkalinity are little affected.

Malarial Pigment.—Ascoli (*Policlinico*, 1910) says that the pigment found in the spleens of individuals dying of pernicious malaria presents the same picture as that seen in the spleens of animals poisoned with pyrodine. The pigment gives the spectrum and chemical reactions of hematin, into which the endoglobular parasite converts the hemoglobin. The hemoglobin content of an infected red blood corpuscle depends upon the variety of the hematozoön which it harbors and not upon the amount of pigment produced. The pigment is formed particularly in the spleen and bone marrow, where the parasite undergoes the last phases of the asexual cycle and a large part of the sexual phases. It persists in the liver and spleen and causes an inflammatory reaction. It disappears quite quickly from the general circulation.

Intestinal Amœbiasis.—The prevalence of amœbic infection of the colon, according to Musgrave (*Philippine Journal of Science*, 1910, v, 229), is of sufficiently frequent occurrence to deserve careful consideration. Fifty fatal cases form the basis of his report. In no one of these was looseness of the bowels a symptom at any time. Indeed, constipation was the feature. At autopsy, however, characteristic amœbic lesions were present in all 50 cases. The lesions varied from those just beginning to those showing ulcers and extensive destruction of the mucous membrane. General symptoms varied greatly, though not pathognomonic, yet strongly suggestive of amœbic infection. Abdominal "aching," more or less general, worse at night and the early morning, and often accompanied by flatulence, is the most frequent symptom. Distention and constipation are common. Loss of weight generally becomes noticeable. Excessive perspiration quite frequently brings the patient to a physcian. Looseness of the bowels, as a dysentery or diarrhœa, has long been the strong diagnostic point, but facts show it also to be a very unreliable one. If the ulceration is low, the sygmoidoscope gives valuable help. There is but one constant finding, and that is the presence of amœbæ in the bowel discharge. Rogers (*Ibid.*, 1910, v, 219) emphasizes the relation of intestinal amœbiasis to tropical liver abscess. In his experience in Calcutta, 57 out of 63 cases of liver abscess have had a clinical history or postmortem evidence of dysentery. In 45 fatal cases of liver abscess, only 1 failed to show the lesions of amœbic dysentery or old scars at the autopsy. In the wards of the European General Hospital at Calcutta, 43 out of 50 cases of liver abscess gave histories of either dysentery or diarrhœa. Finally, the author's experience with ipecac in the presuppurative stage of liver abscess and in the postoperative stage warrants recommendation.

Test for Gastric Cancer.—The glycyltryptophan test of Neubauer and Fischer for cancers of the stomach has already been recorded in this Journal, as well as the modifications in its application suggested by

Lyle and Kober. The test depends upon the presence of a peptid-splitting ferment in the cancerous gastric juice. Incubation of the dipeptid glycyltryptophan and the ferment (the filtered gastric juice) for twenty-four hours results in the production of simple tryptophan, easily demonstrable with bromine water. Neubauer and Fischer emphasized that tryptophan may be present beforehand in the stomach contents; that blood contains a similar ferment and its presence must be ruled out; that trypsin in the stomach juice will accomplish the same cleavage, and bile is indicative of its presence. Lyle and Kober drew attention to variability in results, and required two out of three tests positive before interpreting the results. To avoid having duodenal contents in the stomach, the patients were purged at night, kept upright in the morning, and the test meal given at noon. WEINSTEIN (*Jour. Amer. Med. Assoc.*, 1910, lv, 1085) recommends making use of the ferment's ability to produce tryptophan in the stomach itself. Four or five hours after a meal of bread and butter, meat plainly prepared without extra seasoning or dressing, and some very sweet, weak tea, stomach contents are secured, filtered, and tested with bromine water for tryptophan. If present, the reaction is positive. If absent, the filtrate in a stoppered bottle with or without toluol is incubated for twenty-four to forty-eight hours, and again tested. The most serious defect is inconstancy. If three or four tests are negative, the case is probably non-cancerous. Weinstein does not recommend preservatives. They interfere with a sharp bromine test, and the influence of peptid-splitting bacteria is too slight to need consideration. Blood that is not grossly visible will not contain enough ferment to confuse the results. And the author believes that occult bile could hardly be accompanied by sufficient trypsin to produce a detectable amount of tryptophan in the test. After all is said, one fact stands out prominently: That the test yields positive results in practically all cancer cases and hardly ever in others, as may be seen in the results by Neubauer and Fischer, Lyle and Kober, and Weinstein.

A Comparison of the Results Obtained with the Different Sphygmometers.—AMBLARD (*Archives gén. de méd.*, Paris, 1910, iii, 513) draws attention to the fact that the many instruments for measuring arterial tension are based upon four principal methods, namely: (1) Compression of the artery by an ampulla (Bach); (2) compression by a band (Riva-Rocci); (3) compression of the finger by a ring (Gaertner); and (4) the oscillatory method, indisputably preferable and totally different from the others. The difference in standards for normal individuals depends upon the difference in the instruments, and upon the fact that some measure the maximal arterial pressure, some the minimal, and others the arteriole tension. The sphygmometers of Bach, Potain, Sable, Riva-Rocci, and the sphygmo-signal of Vaquez all indicate maximal pressures. Comparative records show that there is absolutely no parallelism which can be established *a priori;* whereas the normal pressures with one of these methods may be about 30 mm. of mercury higher than those of a second method, in measurements of hyper- or hypotensions no such corresponding relation exists. The differences between the Potain and Riva-Rocci sphygmometers in a normal individual may be from 20 to 30 mm.; in pathological conditions they vary from 10 to 100 mm.

On the other hand, Amblard found that the instruments recording minimal pressures (the sphygmometer of Hill and Barnard, Amblard's sphygmometroscope, Lagrange's pulsocardioscope, and the oscillometer of Pachon) furnish comparable and parallel results. It is impossible to establish a parallelism between the results furnished for the maximal tension by the different methods, which in itself is reason enough against looking for any correlation of these same results with those furnished by other instruments. It is advisable to compare only those results obtained by the same instruments; and the recognition of the one or the other tension is equally indispensable for an appreciation of the conditions of the arterial circulation. Finally, Amblard found that the method of Gaertner, compression of the finger with a ring, gave almost identical results as the Riva-Rocci. It demonstrated that the pressure in the arterioles is practically the same as the maximal tension.

The Influence of the Position of the Body upon Evacuation of the Stomach.—PERUSSIA and MARKOVICZ (*Gazz. med. italiana*, March 31, 1910) demonstrate the value of radiological examination of the motor function of the stomach when the patient is placed in the left and right lateral (prone) positions. For instance, a little difference in the time of evacuation in the two positions, a rapid emptying in both left and right positions, points to hypermobility. In atonic motor insufficiency, the left lateral position retards enormously the time of evacuation, whereas the right accelerates it markedly. The difference in the two positions is very great. In motor insufficiency due to pyloric stenosis, the left position retards the emptying less than it does in atonic insufficiency. The resulting hypertrophy compensates somewhat for the stenosis. But in contrast, the right position does not hasten the evacuation. The cause of the insufficiency, the obstruction, persists. In this case the difference in the two positions is slight. Perussia and Markovicz emphasize the advantage of the right lateral position in cases of atony of the stomach, as well as the disadvantage of the left.

SURGERY.

UNDER THE CHARGE OF

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Circular and Lateral Suture of a Vein.—ZAAIGER (*Zentralbl. f. Chir.*, 1910, xxxvii, 1283) has performed several autoplastic and homoioplastic kidney transplantations in dogs, and has one dog which has lived more than two years with the left kidney transplanted to the inguinal

region and the right kidney extirpated. There are, however, a number of difficulties in the technique which, with biological troubles, cause failures in these experiments. One of these technical difficulties lies in the vein suture. Zaaiger often found at autopsy on the animal that a thrombosis had developed at the vein suture, while the arterial suture was faultless. One of the chief principles of vessel suture is the adaptation of the endothelial surfaces which is to be obtained by turning out the intima as much as possible. This is much more easily done with arteries than with veins. By the methods of Carrell and Stich the retained sutures penetrate the wall of the vessel only once, and after turning out the intima as much as possible they are knotted. Zaaiger, in introducing his retaining sutures from the intimal side into each end of the divided vessel, passes them under the intima and out again as in a transposed Lembert suture. When the knot is tied the intima is spontaneously and easily turned outward, so that the remaining sutures can be introduced without further attention being paid to the turning out of the intima. By this method a circular suture of the vena cava of a dog was carried out and an absolutely ideal result obtained. The following method of applying a lateral suture to a vein was employed in a case of cancer of the penis with adhesion of cancerous nodes to the femoral vein: The adherent mass was drawn upon so that the wall of the vein was brought to a point, where it was ligated. A suitable forceps was then applied on the vessel side of the ligature, approximating the two walls of the vein. On the vessel side of the clamp a suture was passed from one side to the other through the two walls of the vein and tied at both ends. The forceps was then removed and the mass together with the diseased portion of the vein cut away. The part of the vein contused by the forceps was then turned in by a continuous suture. Endothelium was thus approximated to endothelium and the blood stream in the main vessel was uninterrupted. A good result was obtained.

An Explanation of the Pathogenesis of Appendicitis Based upon Experimental and Bacteriological Investigations.—HEILE (*Mitt. a. d. Grenzegeb. d. Med. u. Chir.*, 1910, xxii, 58) has undertaken to study, particularly, why, in so many cases, appendicitis becomes localized, and why, in many cases, it becomes a dangerously progressive inflammation. For several years he has been attempting the solution of the question by bacteriological examinations, but, like others, he has not met with much success. Better results have followed animal experimentation. Simple ligation of the appendix in dogs was followed by the reëstablishment of the canal of the appendix after the suppurative division of the wall and the separation of the constricting silk ligature. If, peripherally to the ligature, a paraffin plug was introduced into the lumen of the appendix, then the peripheral portion remained closed and the canal was not reëstablished. This gave rise to impairment of the health of the animal. It did not cause peritonitis or death, but only a circumscribed inflammatory process, which was followed, after weeks, by adhesions of the omentum and neighboring coils of intestines. The portion of the appendix peripheral to the ligature became more or less dilated and filled with more or less abundant leukocytic contents (hydrops or empyema), usually with numerous bacteria, which, how-

ever, did not produce a progressive inflammation. The tissues of the surrounding wall, on the contrary, showed a slight difference from the normal histological picture, and after a few weeks only here and there a round-cell infiltration. If a portion of the appendix containing normal feces is ligated with silk, and injected with paraffin, then there develops from the feces a chemical and bacteriological infection of the wall, which, very much like the same type of appendicitis in man, proceeds to destruction of the wall and the development of a diffuse, suppurative, ichorous peritonitis and death of the animal, according to the severity of the infection, in from one to five days. The pathologico-histological picture of the destructive inflammation is characterized by two typical courses. In the first there is a localized epithelial defect, which is accompanied by a rapidly progressing suppurative lymph-adenitis of the intestinal wall. The abundantly developed tissue of the lymphoid follicles favors this process very much. In the first few hours after the beginning of the inflammatory process, there develops on the peritoneum, by extension through the lymph paths, an extensive layer of exudate (containing bacteria, leukocytes, and fibrin), although on the mucous surface there can be found only a very small defect. In the second type the excessive development of chemicobacteriological toxins does not result from the primary inflammation and advancing lymphangitis, but from the excessive necrosis, first, of the mucosa, then of the rest of the wall, until perforation occurs into the peritoneal cavity. The course of the process will be favored by the increasing fecal and paraffin contents, because the progressing inflammation is producing exudation in the interior of the lumen and the separation of the portions of the tissues, which adds to the pressure that is to produce the perforation of the badly diseased appendiceal wall. The changes in the blood-vessel walls are of great importance in the course of the destructive inflammation. Either there develops a hyperemia, as from a hemorrhagic infarct, or there occurs an anemic, necrotic infarct. It may be said with certainty that vessel changes are not necessary for the origin of the inflammation. For the origin of the destructive appendicitis, of the very greatest importance, is the retention of feces in the closed end of the appendix. Bacteria alone never produce a destructive inflammation.

Anuria after Gallstone Operations.—CLAIRMONT and HABERER (*Mitt. a. d. Grenzegeb. d. Med. u. Chir.*, 1910, xxii, 159) call attention to cases of severe gallstone disease with common-duct obstruction or cicatricial stenosis, in which, at the conclusion of the operation, there develops dangerous oliguria or fatal anuria. These severe disturbances of the urinary secretion are to be traced to parenchymatous degeneration of the kidneys, which, aggravated by the effect of the damage to the liver parenchyma, are overcome by the influences of the operation and narcosis. A complete anuria is accompanied by an exhaustion of the biliary secretion, due to an inhibition of the liver function. The disturbance of the kidney function is not dependent upon any single factor, such as the long-continued jaundice, alone, but is due to the combination of all the factors resulting from the severity of the disease. Individual predisposition appears to play a certain part. Clairmont and Haberer emphasize that, according to their observations,

all kidney symptoms may be absent, and that in individual cases, from the surgical side, a definite prognosis cannot be given. These accidents in gallstone disease can be provided against only by operation before severe liver disturbances develop. The specific damage to the parenchymatous organs that results from extensive operations, long-continued narcosis, and anemia are to be avoided as far as possible. Conditions entirely similar to those observed clinically in men can be produced experimentally in dogs.

The Mobile Cecum as a Cause of Many Cases of Chronic Appendicitis.—STIERLIN (*Deut. Ztschr. f. Chir.*, 1910, cvi, 407) bases his extensive study of this subject upon 61 cases upon which Wilms did the operation of cecopexy. Twenty-five per cent. were in males and 75 per cent. in females. Sixty-seven per cent. were between the ages of fifteen and twenty-five years. In most of the cases the diagnosis of a mobile cecum was based upon the following symptoms: (1) Periodic attacks of colicky pain, localized chiefly in the region of the cecum and ascending colon, generally without fever, often associated with long-continued pain in this region, and not rarely in the region of the stomach. The attacks of colic occurred in 86 per cent. of the cases. (2) Chronic, usually severe, constipation, occasionally alternating with brief periods of diarrhœa, and particularly at the conclusion of an attack of colic. Severe constipation occurred in 77 per cent. of the cases. (3) In the region of the cecum is a distended tumor, which is distended by gas, and on palpation is soft and gurgling, is painless or somewhat tender, and shows considerable mobility. In all cases the diagnosis was established by the *x*-rays, which determined the abnormal mobility of the cecum, its size and atony, and the diminished motor function. The permanent results in 43 cases were: 75 per cent. cured, 16 per cent. improved, and 9 per cent. unimproved. In 9 cases of long mobile cecum with the typical symptom-complex, appendectomy without cecopexy gave the following results: 2 cured (with persisting constipation), 3 improved, and 5 unimproved. An abnormally long cecum, in rare cases, through kinking and rotation, may lead to severe clinical disturbances. The clinical picture of a mobile cecum develops when, besides a long cecum, there is also a primary or a secondary atony of the colon, or other cause of stoppage in the large intestine, as at the splenic flexure. Atony of the colon or cecum may be congenital, it may be due to a chronic colonic constipation, to an abnormal situation of the colon, or to a combination of these factors. The chronic constipation is most frequently due to a mobile cecum. The colicky attacks of pain and the continued pain in the intervals are due to the pull on the mesentery of the overdistended cecum and, in consequence of the constipation and probably also antiperistalsis, the outward displacement of the cecum. It is probably also due to the effect of the distention on the sensitive nerves in the cecal wall.

Concerning the Value of a Simple Continuous Suture Passing through the Whole Vessel Wall.—SCHILLER and LOBSTEIN (*Deut. Ztschr. f. Chir.*, 1910, cvi, 487) say that a simple continuous suture passed with the aid of retaining sutures is well suited for the anastomosis of vessels, and that the operation is easily carried out. The finest silk and a thin

needle are employed. With the strictest asepsis, favorable results were obtained in 40 per cent. of the arterial sutures and in 60 per cent. of those in veins. In the most severe wounds of the vessels, with loss of vessel substance—up to 2 cm.—by means of a simple circular suture, a favorable result was obtained. When the loss of vessel substance is greater than 2 cm., vessel transplantation is to be preferred. Suture of the smaller arteries gives a relatively better result than of the veins, since in the larger arteries, because of the blood pressure, the suture will be insufficient. It tears out and causes a secondary hemorrhage. Suture of the larger veins gives a better result than of the smaller, since the latter easily thrombose. A completely favorable functional result was obtained in more than 40 per cent. of the cases in which longitudinal vessel wounds were closed by a continuous suture, and in those in which a circular suture was employed after a transverse division of the vessel, as in resections—the loss of vessel substance being less than 2 cm. No aneurysmal formation was observed up to six months after the operation by this simple suture method.

Cancer of the Prostate.—YOUNG (*Ann. d. mal. d. org. gén.-urin.*, 1910, ii, 1742 and 1840) reports an elaborate study of 111 cancers of the prostate, nearly all personal cases, or examined by Young. Two were below fifty years of age; 25 were from fifty to fifty nine-years; 50 from sixty to sixty-nine years; 17 from seventy to seventy-four years; 13 from seventy-five to seventy-nine years; and 4 from eighty to eighty-four years. Carcinoma of the prostate is more frequent than is generally supposed. It was found in 20 per cent. of prostates increased in volume. In 500 cases of urinary obstruction, 400 were due to simple hypertrophy, and 100 to carcinoma. The symptoms were, in general, the same as those of hypertrophy, but the pain, local and radiating, simulates often that of other affections (renal, spinal, and sciatic) and is often the principal symptom. Carcinoma can begin by a small nodule in a prostate otherwise healthy, or one presenting a chronic prostatitis. A simple hypertrophy may become the seat of cancer, but usually in a non-hypertrophied region. It occurs more frequently in the posterior subcapsular region, behind the hypertrophied lateral lobes. Cancer remains localized a long time within the prostate, the ureter, bladder, and posterior capsule resisting invasion a long time. The periprostatic invasion begins generally along the ejaculatory ducts in the space above the prostate, between the seminal vesicles and the bladder and above the aponeurosis of Denonvilliers. The cancer generally reaches the inferior surface of the trigone and the lymphatics which extend toward the walls of the pelvis. The pelvic nodes, however, are involved late, and often the affection attacks the bone before the nodes. Examination of the prostate and seminal vesicles ought always to be practised, and when the prostate is found to be hard or only an indurated nodule in a man past forty-five years of age, cancer ought to be suspected, especially if there is no history of prostatitis, and if there is no hypertrophied lobe projecting into the bladder. In these cases, even if there are few symptoms, an exploratory operation should be done, and if the appearance alone of the posterior surface of the prostate does not suffice for a diagnosis of cancer, it will be necessary to make an immediate examination, microscopically, by the freezing method, of small pieces removed

from the lateral lobes. If the lesions are cancerous, the incision made should be cauterized, and a radical operation performed. A cure can be obtained only by a systematic excision of the seminal vesicles, the vasa deferentia, and the anterior two-thirds of the trigone. The operation is neither difficult nor dangerous, and a permanent cure has been obtained by this procedure (five years elapsing after the operation). When the disease is advanced and urination difficult, it will be necessary to catheterize the patient frequently. When catheterism is difficult or painful, a palliative operation will be necessary; in some cases suprapubic cystotomy. Excellent results, however, in combating the obstruction and vesical pain have been obtained by conservative perineal prostatectomy.

Transthoracic Cardiotomy, a New Method for the Cure of an Impassable Cicatricial Stricture of the Œsophagus.—FISCHER (*Zentralbl. f. Chir.*, 1910, xxxvii, 1393) offers an operation which he says will succeed in those cases in which all other methods of cure have failed. In a boy, aged three years, with an almost impassable stricture of the œsophagus, the stomach was opened for the purpose of performing a gastro-enterostomy, when it was found to be the seat of an hourglass constriction. This was removed by the pyloroplastic operation of Heineke-Mikulicz. A Kader gastrostomy, with Senn's modification, was performed, and to place the stomach at rest a tube was passed through the pylorus into the duodenum. A skiagram taken after the operation showed a stricture of the œsophagus extending from the bifurcation of the trachea to within 2 cm. of the diaphragm. A fine, thread-like shadow showed that the bismuth had passed the stricture. Various attempts to pass the stricture by silk thread and other means from above and below failed, and the child was being nourished poorly through the gastric fistula. Under continuous insufflation of the lung by the Meltzer-Auer method, the thorax was opened in the seventh intercostal space and the ribs separated. As the opening proved to be too high, a second was made in the eighth space, which gave easy access to the diaphragm and lower part of the œsophagus. The lung was retracted by silk cloths sterilized in vaseline. The pleura covering the œsophagus was divided, the vagi were pushed aside, the œsophagus freed from its bed and wrapped in gauze. The œsophagus was then separated from the diaphragm with a blunt forceps and the peritoneum drawn through, and with it the stomach, into the thoracic cavity. The stomach was easily drawn up to about 7 cm. from the entrance of the œsophagus. The pleural cavity was carefully packed off with the silk cloth already mentioned, and the cardia divided between two suture retractors transversely, about 3 cm. below the œsophageal opening. A ureteral catheter with a mandrin was passed easily through the stricture and out of the mouth, where it was secured with an arterial clamp. A suitable forceps was then passed through the gastro-enterostomy opening to the incision in the cardia, where it seized the lower end of the catheter and drew it out of the stomach fistula. The incision in the cardia was closed by silk sutures in a double row, the stomach returned to the abdominal cavity, and the œsophagus fixed to the diaphragm and peritoneum. The thoracic wound was closed and a dressing applied. The patient withstood the operation well, but through a failure in the technique the

patient received an overwhelming and concentrated dose of ether and collapsed. He died twenty-four hours after the operation. Fischer looks upon it as a useful and simple operation.

Saccular Autoplasty in Necrosis of the Intestine from Strangulated Hernia.—LERDA (*Zentralbl. f. Chir.*, 1910, xxxvii, 1339) calls attention to the restricted field for the employment of the omentum in reinforcing sutures in the intestine, or in circumscribing an abdominal organ. The peritoneum has also been used for similar purposes, but not in strangulated herniæ, where it is useful and its technique simple. Often the strangulated portion of the bowel is of doubtful vitality, and is either distinctly necrotic or has already been perforated. Only in the more advanced cases has the gangrene extended to the whole strangulated loop. Generally, the necrosis is limited to the constricted hernial ring, while the remaining portion of the involved bowel is merely the seat of a passive hyperemia. In such cases the choice of operation lies between resection of the suspected intestine, the formation of an artificial anus, or a turning in of the necrotic portion, which is rarely possible, on account of the disturbances in nutrition in the neighboring tissues. In a severe case of strangulated hernia in a woman, aged seventy-four years, Lerda, after so incising the sac as to make a movable flap from it, applied the external surface of this flap over the necrotic band and sutured its free end to the mesenteric attachment of the bowel. An iodoform tampon was introduced into the hernial ring and the wound closed. A fecal fistula resulted. The first stool occurred on the twelfth day, and after that about every second day. On the thirty-fifth day the patient left the hospital with a fistula which would hardly admit a probe. In a second case the same operation was performed, with the exception that instead of the external or bloody surface of the sac, the internal or endothelial surface was applied to the intestine. This patient had a stool on the third day, and regularly afterward. On the sixth day a fecal fistula developed, but it was completely closed twenty-three days after the operation.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Syphilis with "606."—IVERSEN (*Münch. med. Woch.*, 1910, lvii, 1723) substantiates results of other observers with his experience at St. Petersburg with 60 patients. He found the intravenous injections more satisfactory. He says that they are less painful than intramuscular injections and, furthermore, enable a more accurate dosage. In addition their action is more rapid and the dose required is smaller. He says that intravenous injection of "606" is usually followed in two or three hours by a chill lasting half an hour, accom-

panied by a rise of temperature, frequently by pains in the limbs, and in some cases by vomiting and fluid stools. The next day the patient feels well except for some weakness. He found spirochetes constantly in the primary chancres before treatment, but was unable to find any two or three days later. He aspirated fluid from the enlarged inguinal glands and found spirochetes constantly before treatment, but after three or four days after the injection of "606" they could not be found. More superficial lesions retrograded more rapidly. Primary scleroses and adenitis required the longest time, taking three or four weeks to disappear. The Wassermann reaction became negative usually between the twentieth and fortieth day after the injection, and in two cases on the eighth and tenth days. He noticed that the reaction became more intense immediately following the injection, and then gradually subsided. No untoward after-effects were observed.

The Treatment of Syphilis with Ehrlich's "606."—NICHOLS and FORDYCE (*Jour. Amer. Med. Assoc.*, 1910, lv, 1171) believe that we have in arsenobenzol a most thorough agent in controlling the manifestations of syphilis that are caused by the presence of treponema. They say that at present the method recommended by Wechselmann seems to be the preferable one for administering the remedy. They call attention to the fact that delay or insufficiency in the amount of mercury in the early treatment of syphilis permits the entire system to become infected by Treponema pallidum, and damage to the bloodvessels or important structures takes place which might be prevented by energetic treatment. Although mercury and potassium iodide are efficient in the great majority of cases in controlling the manifestations of the disease, there are certain intractable cases in which they fail to control the symptoms, either on account of idiosyncrasy to one or both of these drugs or other causes. Furthermore, owing to the length of time required in treating a case of this infection patients often become discouraged and cease medication after a few months or a year or two. It is also well known that there are certain cutaneous and general manifestations of syphilis which respond slowly or not at all to mercury, even in heroic doses. Among these may be mentioned certain scaling syphilides of the palms, chronic nodular syphilides in patches of the type which clinically resembles lupus vulgaris, certain forms which affect the flush area of the face and bear a marked clinical resemblance to lupus erythematosus; also mucous membrane affections like leukoplakia, chronic interstitial glossitis, and relapsing deep and superficial lesions of the tongue and throat. Aside from these lesions which are under direct observation, there are the so-called parasyphilitic manifestations, such as tabes, the treatment of which by the classical drugs leaves much to be desired. In malignant syphilis with early destructive lesions and the development of profound cachexia, mercury, moreover, often fails to do good and not infrequently does harm. They conclude that it may reasonably be hoped that all the lesions which depend upon the presence of the organism will be favorably influenced, and the most we can expect in the secondary degenerative changes is that the process may become arrested. Further experiences with the drug will determine with more accuracy the dose which is necessary to bring

about a cure, the time that must elapse before a second dose can be safely given, and the more definite indications for its use after relapses or failure of a single dose to control the symptoms.

The Subcutaneous Use of "606."—Michaelis (*Berl. klin. Woch.*, 1910, xlvii, 1531) prefers the subcutaneous injection of this remedy, and says that he has found it well borne without exceptions. Often there was severe pain for some hours after the injection, but this was controlled by moist compresses. Sometimes a transient pain appeared again on the third day, but usually there was absolutely no sign of any inflammatory reaction. The great advantage presented by the subcutaneous method over the intramuscular injection is that walking and sitting are not interfered with by a dense infiltrate. The action of the remedy is more rapid than when it is given intramuscularly because of the greater surface exposed to resorption. Michaelis states that he has not seen any untoward by-effects in 71 cases of syphilis treated with Ehrlich's "606."

The Treatment of Sciatica with Epidural Injections.—Blum (*Münch. med. Woch.*, 1910, lvii, 1681) refers by the term epidural injections to the anesthetization of the sacral nerve roots after they have left the cord, a method discovered simultaneously by Cathelin and Sècard. Blum has also used the perineural technique, and compares the two methods. He found that epidural injections were efficacious when the perineural injections failed. He injects from 5 to 10 c.c. into the spinal canal through the lower median sacral foramen, the needle piercing only the sacrococcygeal ligament. He says that the epidural method is not painful and has never caused fever or other untoward effects. On the other hand, injections by the perineural method have produced paralysis of the peroneus. Furthermore, epidural injections affect a wider nerve distribution than do perineural injections. Thus, the application of epidural injections is much wider, and they have been successfully used to relieve pain from compression of nerve roots by cancer of the spine, to relieve the gastric and bladder crises in tabes, and to relieve pain in lead colic. Furthermore, they have also been used for the treatment of nocturnal enuresis. Blum gives the details of the technique in his article which should be consulted in the original.

Chlorosis.—Morawitz (*Münch. med. Woch.*, 1910, lvii, 1425) forms the following conclusions as a result of his studies of chlorosis: (1) There are marked cases of chlorosis in which the condition of the blood is normal or nearly so. Such cases are not rare. The influence of iron is just as favorable in these cases as in those associated with distinct anemia. (2) Anemia is therefore not the cardinal symptom of chlorosis, but only one symptom among others. (3) The disturbances of menstruation, venous murmurs, and the majority of the subjective symptoms are not always dependent upon an anemia. (4) It is improbable that the curative action of iron in chlorosis is to be ascribed to a stimulative effect upon the formation of hemoglobin. The inferior action of iron in almost all non-chlorotic anemias, as compared with arsenic, aside from other observations, is an evidence against the existence of such a stimulative action. (5) Iron has a beneficial action in chlorosis not alone

because of its action upon the blood-forming organs, but also because of a direct action upon the, as yet unknown, cause of the disease. This, Morawitz suggests, may be a defective or excessive functioning of the ovaries or some interrelated ductless gland. The fact that chlorosis develops exclusively during puberty is also suggestive of this relation. He cites the observations of other men who have similar views, and gives the details of some cases he has observed. In these cases the percentage of hemoglobin was above 80 per cent. in nearly all, not below 60 per cent. in any, and frequently over 90 per cent. Morawitz does not believe that the mere loss and recovery of the difference between 80 and 90 per cent. of hemoglobin could either produce or cure the train of symptoms that make up the disease chlorosis.

The Present Status of Antityphoid Inoculation.—GOSMAN (*Jour. Amer. Med. Assoc.*, 1910, lv, 1169) gives a brief resume of the literature on inoculation for typhoid fever and the preparation of the vaccine and the methods of its use. In concluding, he says that it seems that the present status of these inoculations against typhoid fever is that they are valuable as a method of preventing the disease, and are, perhaps, the most valuable single asset we have in combating an epidemic, and that there is surely no doubt in the minds of most thinking and up-to date medical men that they should be used in the following classes of persons: First, all nurses, ward attendants, hospital corps men, Red Cross assistants, physicians, and medical students; also all persons who contemplate a journey into a section where typhoid fever is known to exist or is suspected of existing. The inoculations should also be done generally in districts suffering from an epidemic, and especially in the families where a case exists; and in time of war all volunteers at camps of concentration should be inoculated as soon as possible after the camp is started. Gosman is convinced of the harmlessness and, at the same time, of the effectiveness of this procedure.

The Influence of Age and Temperature on the Potency of Antidiphtheritic Serum and Antitoxin Globulin Solution.—ANDERSON (*Jour. Infect. Dis.*, 1910, vii, 481) reports his work undertaken to determine the effect of age and temperature on the potency of diphtheria antitoxin. He tested both the untreated horse serum and the globulin preparation as made by the Gibson process. Each sample of serum was divided into three portions; one portion was kept at room temperature, one at 60° F., and the other at 40° F. The potency of each sample was determined every six months for three years. He found that the average yearly loss in potency of diphtheria antitoxin at room temperature was about 20 per cent.; at 60° F., about 10 per cent.; and at 40° F., about 6 per cent. He could determine little difference in the keeping properties of the untreated serum and the globulin preparations. He believes that if allowance is made for the decrease in potency, old serums are just as efficacious for therapeutic purposes as fresh serums. Diphtheria antitoxin to be placed upon the market and then kept under unknown conditions as regards temperature should be labelled with a return date not longer than ten years, and should contain an excess of at least 33 per cent. to allow for decrease in potency; in addition, when the serum is sold in syringes with an absorbable piston, an excess should be added for this loss. The only

disadvantage in using old serums would be the increased bulk of the injections for a given dose. Anderson also found that diphtheria antitoxin, if dried and kept in the dark at 40° F., retained its potency practically unimpaired for five and one-half years. He suggests that such a dried antitoxin might be of value on long voyages or in the tropics.

Deep Perineural Injections for the Relief of Neuralgia and Sciatica.— HECHT (*Med. Record*, 1910, lxxvii, 1040) reports his results in 60 cases of trifacial neuralgia. He used alcohol in ascending strengths of 70, 80, and 90 per cent. for the injections. Of the 60 patients, 37 were distinctly benefited (complete abeyance of pain for a longer or shorter period of time); 11 were improved (great reduction of pain); 8 were unimproveed, and in 4 patients the pain was aggravated. Of the 37 patients benefited, 1 patient was entirely free from pain for one year and eight months. The shortest free interval from pain was two weeks. Hecht says this variability in results cannot be satisfactorily explained, but may be due to the accuracy with which the alcohol was deposited in, at, or near the nerve. With regard to the injection treatment of sciatica, Hecht says that alcohol injections are decidedly contraindicated. Harmful and even disastrous results have followed alcohol injections into mixed nerves. He reports a series of 33 cases of sciatica treated by the injection method. This method of treatment is limited to those cases in which the diagnosis of sciatica is made only after a rigid exclusion of all conditions that may give rise to sciatic pain as a symptom. Among the conditions that may give origin to pain similar to sciatica he mentions arthritis of the rheumatoid or deformans variety, tuberculous spondylitis, tabes, spinal syphilis, diabetes, inflammatory pelvic exudates, intermittent claudication, myalgia, and advanced general arteriosclerosis. He used deep intraneural injections of salt solution, and found that in most instances the relief from pain was almost immediate and complete. There were neither unpleasant effects nor any complications following the injections. Hecht also found that these injections benefited cases that may be classified from the site and distribution of the pain as myalgia.

The Action of Cod-liver Oil in Rachitis and Spasmodic Affections of Children.— ROSENSTERN (*Berlin. klin. Woch.*, 1910, xlvii, 822) reports his clinical experience that seems to confirm Schabad's conclusions with regard to the specific action of cod-liver oil upon the metabolism in rachitis and the convulsions so often associated with rachitis. Rosenstern gives the details of five cases of rachitis with marked craniotabes. In all these cases remarkable benefit followed the daily administration of 25 grams of cod-liver oil for three to six weeks. He thinks that phosphorus enhances the action of the cod-liver oil and advises the use of a mixture of 0.01 part of phosphorus to 250 parts of cod-liver oil. Of this mixture a teaspoonful is given five times a day to the severest cases. Cod-liver oil if given without phosphorus will have the same effect, but must be given in larger doses.

PEDIATRICS.

UNDER THE CHARGE OF
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The Diagnosis of Nasal Diphtheria in the Newborn and in Infants.—BLOCHMANN (*Berl. klin. Woch.*, 1910, xlvii, 2008) claims that nasal diphtheria in infants, in a large percentage of cases, is mistaken during the first days and even the first weeks of the disease for the ordinary "snuffles" or coryza. Notwithstanding the fact that this condition is frequent in infancy, and that the child's appearance is characteristic to the experienced eye, the true diagnosis is often not made until a rapid loss of weight and strength, a diphtheritic otitis media, or an absolute occlusion of the nasal passages make their appearance. It is, therefore, important to make a diagnosis immediately by direct nasal examination, before receiving the bacteriological report. It is desirable to be able to diagnosticate the bacilli carriers from those ill with the disease. Direct nasal inspection is remarkably easy in the newborn. In the recumbent position, with the child's nostrils separated by a bent hairpin or an open speculum, a good view of the nasal passages is obtained. If the child is not crying, pressing backward the tip of the nose is often sufficient. These methods were used in two epidemics among families. Three cases were diagnosticated as nasal diphtheria previous to a bacteriological report out of 8 cases of coryza. The second epidemic showed 5 infants, in their first week, infected by an adult. The first 2 cases showed coryza, and were diagnosticated diphtheritic by the direct nasal examination. This method then showed 3 other cases out of 16 children in the institution. These diagnoses were subsequently proved correct by cultures, and none of the remaining infants with coryza gave positive cultures or developed diphtheria. Among these 10 cases of nasal diphtheria, but 1 showed the characteristic discharge of bloody serum from the nostrils; 5 showed a thin membrane on the septum, which seems a rather frequent location. In only 1 case was the nasal floor involved. In a few cases the membrane involved the turbinates. The first epidemic developed in older infants, and was of the chronic type, almost always without fever. The membrane persisted in one case for over four weeks, and in another case the cultures were positive for three months. In the second epidemic 2 of the infants also developed diphtheritic inflammation of the navel, one dying of peritonitis. All the other cases recovered under antitoxin treatment. Only one child exhibited a rise in temperature. The foregoing history shows that rhinoscopic examination of the anterior fossæ is practicable in the newborn, and is an efficient method of preventing a dangerous neglect in diagnosis and delay in proper treatment.

Immunized Human Serum in Hereditary Syphilis.—MEIROWSKY and HARTMANN (*Medizinische Klinik*, 1910, vii, 1572) have investigated the effect of serum taken from syphilitics recently treated by "606" on the

lesions of hereditary syphilis in an infant. The good results obtained by treating nursing syphilitic mothers with "606," and influencing the child's condition through the mother's milk, led them to try the effect of the serum. The child of a syphilitic mother developed typical lesions of hereditary syphilis two weeks after birth. The lesions consisted of a pemphigus-like eruption on the soles of the feet, the palms and dorsum of the hands, and the buttocks. There were circumscribed areas of efflorescence around the mouth, on the ankles, and some around the buttocks. The mother's milk having failed, advantage was taken of the period to employ hypodermic injections of the human serum. The injections were as follows: September 3, 20 c.c.; September 6, 18 c.c.; September 7, 8 c.c.; September 9, 5 c.c.; September 10, 2 c.c.; September 11, 5 c.c.; September 14, 10 c.c.; September, 16, 18 c.c. Two days after the first injection the soles of the feet were clear, the lesions on the hands much improved, and the efflorescence paling and showing a slight desquamation. The lesions continued to diminish, and on September 17 all the lesions had disappeared except a slight superficial desquamation at one place. Three days after the last injection there developed a periostitis of the right arm. Although not an entirely absolute cure, yet the efflorescence disappeared after the first injection, and the skin lesions which were progressive were halted and cured. The authors believe the effect of the injections would have been still more marked if they had had larger doses of the serum to use.

The Diagnosis of Epidemic Meningitis in Infants.—Ernst Levy (*Medizinische Klinik*, 1910, vi, 1569) points out that the characteristic symptoms of acute onset, convulsions, and high fever, and the classical signs of rigidity of the neck and Kernig's sign do not usually appear in the meningitis of very young infants, excepting very late in the disease. Kernig's sign and rigidity of the neck are absent in from 50 per cent. to 60 per cent. of the cases under two years of age. Convulsions, which occur in so many infantile diseases, especially in spasmophilics, occur in young children with meningitis in only one-third of the cases. Intestinal irritation without fever and also inflammation of the entire respiratory tract often occur for weeks before some other definite symptom which indicates meningitis is found. Infants are especially liable to secondary infection of the lungs in meningitis, the true nature of which is thus often masked. With a few exceptions, almost all cases of meningitis in children of two years and under, Levy finds, have been treated for a variable length of time, even forty-five days, as cases of intestinal or pulmonary catarrh. Rigidity of the neck alone is a slight but fairly frequent accompaniment of these inflammations of the lung or intestine. A remittent type of fever is rather characteristic, the periods between the rises being short at first, and grow longer as the disease advances. There is often no febrile reaction at all. Levy has not found slowing of the pulse a characteristic feature, except in some forms of tuberculous meningitis. Symptoms referable to the central nervous system, such as the patellar reflex, etc., are, unfortunately, usually absent or doubtful. However, strabismus, usually fleeting, and pupillary inequalities are among the most constant signs of the disease. Hypersensitiveness, especially of the legs, is a cardinal symptom, as also are the pupillary dilatation from pain, on pinching the spine, and a reflex tremor of the

whole body or of a group of muscles on sitting the child erect or moving its limbs. This tremor, however, usually presents itself during or after the fourth week of the disease. Highly important from the diagnostic viewpoint are alterations in sight or hearing, such as blindness or deafness, following an illness of uncertain nature. Subsequent to such an illness, otitis media, neuroretinitis, or iridochoroiditis invariably means meningitis. In infants, a widening of the fontanelles is of great diagnostic value, as showing increased tension of the cerebral fluid. This is often an early sign and a common condition in epidemic meningitis. Ventricular puncture is important in diagnosticating all cases showing increased tension of the cerebral fluid, as the cocci are often found here when they are absent in the spinal fluid. Lumbar puncture is essential; but as this means is not always available, and a bacteriological examination requires time, a provisional diagnosis at least should be made from the symptom complex, so that treatment by serum injection may not be delayed.

An Epidemic of Anterior Poliomyelitis in Austria.—GEORG STIEFLER (*Medizinische Klinik*, 1910, vi, 1743) reviews the history of anterior poliomyelitis in Europe, America, and Australia, and notes the different aspects in symptomatology, after-effects, and mortality shown by different epidemics. The clinical picture constantly changes in the different epidemics. In some, gastro-enteric symptoms predominate, in others they are absent. Some epidemics show apparently all forms —abortive, intestinal, spinal, bulbar, meningitic, etc. Mortalities range from 10 per cent. to 22 per cent. The constant and characteristic symptom in all of them is the paralysis with the acute degenerative changes in the central nervous system. Distribution of the epidemics among the summer and fall months is constant. A consensus of opinion has developed that the disease is infectious. That it is spread by contact with infected individuals seems highly probable. That it can be carried by a third, healthy person is doubtful at present writing. Stiefler particularly reports the epidemic in northern Austria in 1909. Most of the thickly settled and industrial communities escaped the disease. Most of the cases occurred among small landowners and laborers, but numbers of the well-to-do also were affected. The greatest number of cases occurred in children aged from one to three years. A relatively high proportion occurred in children aged from six to fourteen years, and 15 per cent. occurred in adults. The disease was prevalent from August to January, with the largest number of cases in October. The clinical aspect showed usually a rather short prodromal stage with variable symptoms. Besides the usual prostration, anorexia, pain in the head, neck, back, and limbs, there was frequently seen an angina and a respiratory catarrh. Gastro-intestinal symptoms, such as nausea, vomiting, constipation, or, less frequently, diarrhœa, were often seen. The paralysis was always of the atrophic, degenerative type, reaching its maximum within a few days, then receding and persisting only in certain groups of muscles. The meningitic type rarely appeared, and only twice were the bladder and rectum involved. Pain was present in most cases; sometimes it was general, sometimes limited to the paralyzed muscles. Tenderness on pressure was common in the muscles and nerve trunks. The fever was moderate throughout, and lasted but a

few days. The abortive type was rare. Complete recovery occurred in 22 per cent. of the cases. Most of the other cases recovered with physical defects. The mortality was 12.99 per cent. The pathological process is not always limited to the anterior horns, but may affect the posterior horns and the gray matter of the brain or cord. The disease attacked two or more members of the same family in seven instances, the second case appearing from two to twelve days after the first case developed. In a number of cases the disease showed a decidedly contagious character.

OBSTETRICS.

UNDER THE CHARGE OF
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Pernicious Nausea of Pregnancy Treated by Thyreoidin.—SIEGMUND (*Zntralbl. f. Gynäk.*, 1910, No. 42) reports 5 cases of pernicious nausea of pregnancy, in which he has had good results by the administration of thyreoidin. Four of these cases were under his immediate observation, and one was treated by a colleague. The remedy is administered several hours before the time when the worst paroxysms of vomiting appear. The stomach must be absolutely empty at the time of the administration. It is usually best to give it in the early morning before the patient leaves her bed. She is encouraged to sleep afterward and to take nourishment before getting up. From an hour to half an hour, however, before the other meals of the day, the remedy should be used, and also before going to bed. The morning dose should be the largest, 6 decigrams being given in some cases. If the patient vomits in the evening, a similar dose should be given before this time. The remedy is best used in powder, and sometimes sodium bicarbonate or bismuth may be combined with it to advantage. In other cases, fresh glycerin extract of the gland is used to advantage. In most severe cases Merck's preparation for hypodermic injection may be administered with prompt results. If the vomiting can be controlled the patient should be fed with small quantities of easily digested food, usually liquid. Meat should be avoided. An examination of the urine of these patients showed no disturbance from the remedy, and no bad symptoms developed during its use. Bossi's experiments of giving such cases suprarenal extract was also tried by the author, and in some cases with apparently good results. It has also been observed that in some patients in which the secretion of milk is deficient, the administration of thyroid extract is of decided value.

The Tetany of Pregnancy.—FRANK (*Monatss. f. Geburt. u. Gynäk.*, 1910, xxxii, Heft 4) reports 5 cases of tetany complicating pregnancy. They presented the usual symptoms of muscular cramp in anemic and depleted patients. The fifth case was especially interesting and

typical. The patient was pregnant for the sixteenth time. She had given birth to twelve children, who had died, three remaining alive. In her last pregnancy she had central placenta prævia, and was delivered by version and extraction, with severe hemorrhage. For two months she had had cramps in the hands and in the muscles of the face, which were exceedingly painful, and were not relieved by medication. Recently these cramps became almost continuous. On admission to the hospital the uterus was two fingers' breadth above the umbilicus, and the patient's muscular cramps extended to the upper and lower extremities, and occasionally to the muscles of respiration. When this happened the patient became partially unconscious. The cramps were so severe that full doses of chloral, bromide, and morphine, were given with but very little relief. Labor was induced, after which the patient was considerably better for a time. In the sixth day of the puerperal period she was suddenly taken with fever and a return of the convulsions. This gradually subsided, and the patient made a good recovery. In discussing the causation of the disease, it seems to be a toxemia occasioned by the failure of the immunizing substances in the body to neutralize the toxin produced in the placenta. Others think that the patient produces in her own body so great an amount of toxin that the epithelia of the body cannot neutralize it. In most cases the disease returns in succeeding pregnancies, while the patient is absolutely free when not in the pregnant condition. Multiparæ are most often attacked, and, as a rule, only nervous and anemic primiparæ. The symptoms are most pronounced in the latter half of pregnancy. In each succeeding pregnancy the disease is worse, the cramps gradually attacking not only the upper extremities, but the various groups of muscles throughout the body. Rapidly succeeding pregnancy makes the disease especially severe. The occurrence of hemorrhage in a previous pregnancy predisposes to the disease in the next pregnancy. Meinert reports the case of a patient from whom a goitre was removed in the fourth month of gestation; three days after operation she had tetany, which disappeared in two weeks. The labor was normal. A year later the patient again became pregnant, when tetany returned with such severity that it was necessary to interrupt the pregnancy. As a rule, the cases proceed to recovery. Many mild cases do not come to the attention of a physician. The treatment consists, in mild cases, in rest, forced nutrition, and the use of bromides. In severe cases the pregnancy must often be interrupted when tetany follows extirpation of the thyroid gland. Schauta, who has seen large numbers of these cases, does not believe that when the thyroid gland has not been removed tetany is sufficient reason for inducing labor.

Combined External and Vaginal Version.—Stowe (*Amer. Jour. Obst.*, October, 1910) reports 13 cases of combined external and vaginal version. His experience with these leads him to conclude that combined external and vaginal version is not dependent upon the size of the cervix nor the degree of its effacement. The less the cervix is dilated the more easy the operation, if the membranes are unruptured. During the latter weeks of pregnancy it is often impossible to correct a malpresentation by external methods only. A preliminary dilatation of the perineum in primiparæ, and the performance of this combined

version, does not tend to interrupt pregnancy. In none of these cases did the fœtus, when turned resume its abnormal position. No evidence of separation of the placenta was observed, and in no case were the heart-sounds of the child altered. The proper presentation of the fœtus by the cortex or breech, should be obtained by this method. The operator may choose in accordance with the conditions present. Should amniotic liquid escape during the first stage, version should be done as soon as possible. The danger of separation of the placenta depends upon the degree of uterine contraction and the amount of amniotic liquid present. If the hand does not enter the uterus septic infection rarely occurs. In some cases of placenta prævia the foot can be brought down to the inlet before the membranes are ruptured. This is easier than the classic Braxton-Hicks method.

The Treatment of Fibroids Complicating Pregnancy.—TATE (*Brit. Med. Jour.*, October 22, 1910) draws attention to the unfavorable changes which may occur in fibroids during pregnancy. He believes that should a patient having a very large fibroid become pregnant, serious symptoms may develop in the latter half of pregnancy from upward pressure on the diaphragm caused by the rapid increase in the size of the uterus. Necrosis in the tumor may cause discomfort or persistent pain and make interference with the pregnancy necessary. Torsion of the pedicle in a fibroid tumor is so rare that it need not be regarded as a practical danger. Small fibroids above the brim of the pelvis do not, as a rule, undergo important changes during pregnancy. It is estimated that from 20 to 25 per cent. of women having fibroids abort because of the presence of the tumor. Retention of a portion of placenta and septic infection are not uncommon in these cases, and may necessitate hysterectomy. During labor a fibroid tumor may become wedged in the pelvis, making vaginal delivery impossible. This accident should have been anticipated if the patient is seen in pregnancy and examined, when it is usually possible to dislodge the tumor into the abdomen before labor begins. Occasionally a fibroid uterus may rupture during labor because the uterine wall has become thinned over the tumor. Cases in which hysterectomy is imperative during labor are not very common, and this operation may usually be made elective by an examination of such patients during pregnancy. During the puerperal period postpartum hemorrhage must be anticipated and treated. It may be difficult to control this, because the tumor may become partially dislodged from its bed and great difficulty be found in controlling the discharge of blood from the uterine sinuses. Fibroid tumors may also become detached and partially extruded during the puerperal period; and if sloughing occurs the patient is exposed to the danger of septic infection. When fibroids are situated in the pelvis, and must necessarily obstruct labor, if the tumor is mobile it is usually possible under anesthesia to push it above the pelvic brim and allow the pregnancy to go on. If the tumor is firmly impacted in the pelvis, it is safer to perform abdominal section, with the hope of enucleating the tumor from the uterine wall. Small fibroid polyps attached to the cervix should be let alone if possible, as interference tends to bring on abortion and hemorrhage. When fibroids are multiple and encroach upon the lower uterine segment, it is impossible to

predict with certainty the behavior of the tumor during labor. Such tumors often change their position during parturition, rising gradually into the abdomen as the child descends. When fibroids are entirely abdominal tumors, severe symptoms are rare and operative interference is seldom necessary. The greater number can be treated by myomectomy successfully. Interstitial fibroids rarely justify interference during pregnancy, and should operation be done it is usually best to remove the uterus. If possible, an operation should be deferred until full term, and even then myomectomy may be selected, if possible. The induction of abortion or premature labor should not be undertaken in cases complicated by fibroids because the risk of hemorrhage and septic infection are too great. In view of the fact that many of these patients have but one pregnancy, it is well to take every conservative measure to bring that pregnancy to a successful termination.

Lockyer (*Ibid.*) reports in detail 9 cases of fibroid tumors of the uterus complicating pregnancy. In 3 of these the tumor was incarcerated in the pelvis; in 4 of them operation was performed, followed by uninterrupted recovery; in 3 the uterus was removed; and in 1 the tumor was removed and also a right-sided ovarian cyst. This patient's pregnancy was not interrupted and she had a spontaneous delivery afterward. In 5 cases operation was not done, as the tumor was not in a position to become incarcerated or to be dislodged under anesthesia. In 1 of the cases abortion occurred, and difficulty was experienced in removing the placenta.

GYNECOLOGY.

UNDER THE CHARGE OF

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An Improved and Perfected Operation for the Relief of Extreme Cases of Procidentia, Cystocele, and Rectocele.—GOFFE (*Amer. Jour. of Obstet.*, 1910, lxii, 611) claims that when the uterus and bladder are in their normal position the pelvic floor exercises no influence whatever in their support, which is obtained only by the suspension of their ligaments to the bony framework. The association of prolapse and relaxation of the outlet is due not to the loss of the support as afforded by the perineum, but to the introduction of a new force—the rectocele—which exerts traction on the cervix to which the ligamentous attachments must eventually succumb, permitting the uterus to assume the varying degrees of prolapse. The reason for absence of prolapse in complete tear is that no pull is exerted on the cervix. Repair of the perineum is essential to the treatment of these conditions in order to eliminate the drag of the rectocele. The bladder is likewise held in position by suspension. One support is the uterus, to which the bladder and upper end of the vagina are intimately attached; a second support is afforded by the fascia lata, which, coming under the bladder from its lateral insertion,

acts like a sling. Cystocele, therefore, is due to a descent of the upper support of the bladder—the uterus—and the yielding of the vaginal sheath, permitting a hernia of the bladder. An additional factor is sliding of the various planes of tissue composing the vesicovaginal septum. Based upon these conceptions of production, the author has devised operative procedures to meet the conditions presented. Briefly summarized, the operation for cystocele consists, first, in a free dissection of the bladder from its attachments. After suspending the uterus by shortening the ligaments, the bladder is restored to its normal position and held in place by three catgut sutures, one at its fundus and one at each cornu, thus spreading out and fixing the base of the bladder. After excising the excess of tissue, the fascial sheath and vaginal mucosa are sutured. In case of extreme procidentia, a vaginal hysterectomy is performed and the broad ligaments are sutured together across the pelvis, taking up all slack necessary to make them taut. Upon this support the bladder is spread and sutured, as previously mentioned. The upper end of the vagina is also attached to the broad ligaments after removing sufficient tissue to obtain the support of the fascia lata. In all cases in which a rectocele exists the perineum is repaired.

The Radical Cure of Hemorrhoids: Modified Whitehead's Operation.—By omitting dilatation of the sphincter, O'Conor (*Lancet*, 1910, clxxix, 946) has found that the operation for hemorrhoids can be easily performed in a short time with but little loss of blood. He emphasizes the importance of removing only the thin cuff of mucous membrane along with the loose areolar tissue which bears the hemorrhoidal veins, of carefully avoiding injury to the sphincter by using the senses of sight and touch, and not encroaching too much on the skin when making the primary circular incision. Four Lane's forceps are applied to the four cardinal points of the anal circle exactly at the mucocutaneous junction, irrespective of rugosities. With scissors each segment is divided at the mucodermal line until the circumference has been completed, the primary cut including skin and some subcutaneous tissue. The left index finger is inserted into the anal canal and the separation carried down to the submucosa; the external sphincter is pushed upward with a blunt dissector. No further advance must be made until the submucosa is well defined throughout the whole circumference, and the sphincter has been freed. During the process of freeing the mucosa, forceps are applied to its cut edge to serve as tractors. By blunt dissection the cuff is freed as high as the internal sphincter, which is indicated by the transverse fold of mucous membrane. A vertical slit is made in the mucosa up to this point and the apex sutured to the skin; the cuff is excised by a series of transverse cuts, uniting the mucosa and skin with a continuous catgut suture as the division progresses. Fibrous strictures resulted in 2 out of 450 cases thus treated; a partial recurrence occurred in one patient, who subsequently developed cirrhosis of the liver.

The Wertherim-Schauta Operation.—Based upon his experience in 40 cases, Stoeckel (*Archiv f. Gynäk.*, 1910, xci, 546) details the important points in operative technique as well as a few of the complications which he has observed during convalescence. The operation is used in cases

of cystocele, and consists in the interposition of the uterus between the base of the bladder and the anterior vaginal wall. Of great importance is complete mobilization of the cystocele sack. Too frequently the bladder is separated from the anterior cervical wall only sufficiently to permit delivery of the uterus through the uterovesical fold of peritoneum, which produces an elevation of the centre of the cystocele, leaving unaltered a pouch on either side. Total recurrence is certain to follow, due to the constant traction exerted by these lateral recesses. Complete mobilization of the cystocele requires division of the so-called "bladder pillars," which consist of dense tissue extending from the base of the bladder to the anterior vaginal wall. To avoid hemorrhage, these ligaments should be divided between ligatures. Injury to the ureters will be obviated by incising the pillars transversely close to the vaginal wall at the lower angle of the colporrhaphy wound. Secondly, the uterus must be placed in such a position that it supports the whole bladder. Every uterus which is brought in the position of forced anteversion has the tendency to upright itself. Should this tendency be marked, the fundus will project into the bladder, leaving unsupported a portion anterior to it; or there may be a complete or partial separation of the uterus from its points of fixation. Should force be necessary to bring the uterus under the urethra, the interposition is contraindicated. When the uterus is too small to serve as a proper support, a broad base is obtained by suturing the round ligaments to the vaginal wall. Enlargement of the uterus due to chronic metritis is especially adaptable for this purpose. The cervix is amputated only when it forms a sharp angle with the body of the uterus. In every case the uterine cavity is examined by incising the anterior wall from the fundus to the internal os; after thorough cauterization, this incision is closed. As the last important point, Stoeckel urges that complete hemostasis be secured. Bladder symptoms of varying intensity frequently follow the operation. In one case dribbling of urine developed a few weeks after operation, due to a sacculation of the bladder immediately posterior to the urethra, which resulted from spontaneous elevation of the uterus; a second case, in which the bladder was accidentally opened, developed a stubborn cystitis, which was aggravated by the distortion of the bladder incident to operation. One death resulted from pulmonary embolism. The only disadvantages to this operation are the length of time necessary for its performance, and the increased disposition to thrombosis. Especial care is advisable in the presence of a demonstrable myocarditis.

The Structure of the Stroma of the Endometrium and its Bearing on the Menstrual Changes.—As the result of his studies, YOUNG (*Brit. Med. Jour.*, 1910, ii, 1214) states the following conclusions: The stroma of the endometrium consists of a soft semifluid protoplasmic mass, imperfectly differentiated into cellular elements. The cells anastomose freely with one another by means of protoplasmic processes. They present many and varying alterations in shape, but these are easily dispelled, and the cells then approximate to the typical stellate shape. The differentiation of the stroma cells is thus probably more apparent than real. The intercellular spaces, in all probability, do not directly communicate with one another. The anastomosing processes

are not, as is usually stated, filaments, but films of protoplasm, which under ordinary circumstances close in the fluid cavities. The intima and media of the vessels are nothing more than ordinary flattened stroma cells. This shape they easily lose. Except in the deepest layers of the mucosa, the vessels have no specialized supporting coats (muscle, elastic tissue). The vessels are obviously so constructed as to allow a ready and universal opening up of their walls, and the structure and consistence of the stroma such as to permit its ready displacement by fluid or blood. The œdematous infiltration of the tissues, which precedes the hemorrhagic escape, is due neither to a mechanical displacement or filtration of fluid from the vessels, nor to a secretory activity of the intimal cells. It is dependent on protoplasmic changes, which result in an active imbibition of fluid from the vessels by a process of osmosis. So far as we at present know, this change is due to a widespread liberation of crystalloid elements in the tissues. The infiltration of the stroma with blood corpuscles is, in all probability, due to exactly the same cause. In consequence of its peculiar structure, the uterine mucosa must be looked upon, throughout its whole extent, as a potential blood sponge.

OPHTHALMOLOGY.

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PROFESSOR OF DISEASES OF THE EYE IN THE PHILADELPHIA POLYCLINIC.

Self-inflicted Ocular Traumatisms among Russian Conscripts.—SEIDENMANN (*Centralbl. f. prak. Augenhk.*, October, 1910, p. 293) finds that such injuries, usually involving the right eye, and either self-inflicted or betraying the unskilled handiwork of the quack, may be comprised under three categories: (*a*) Opacities of the cornea opposite the pupil; (*b*) traumatic cataracts; and (*c*) destruction of the entire globe through application of leeches to the cornea. The mechanism producing the corneal injuries is not entirely certain. It is probable that that membrane is first cauterized with nitrate of silver and the wound thus caused treated with potassium iodide. The cataracts always betray their origin by the minute corneal opacities which gave entrance to the needle. The leeches, the cruellest method of all, suck out the aqueous humor, causing extensive intra-ocular hemorrhage, with detachment of the retina and choroid. The final result is a shrunken stump.

Pigmentation of the Retina in Nephritis.—RAGHLIS (*Klin. Monatsbl. f. Augenhk.*, September, 1910, p. 322) from microscopic examination. concludes that pigmentation of the retina in nephritis may occur from degenerative changes of the external and middle layers of that

membrane, the choriocapillaris being intact. Such degenerative changes of the retina can be regarded as consequences of the nephritis. They are not analagous to the pigmentation which occurs in the fatty cells in fatty infiltration of the retina. The ophthalmoscopic appearances of choroiditis disseminata are sometimes found in changes of the pigment epithelium and retina alone when the choroid itself is healthy.

Isolated Monolateral Paralysis of the Third Nerve of Aneurysmal Origin.—From an analysis of cases of this kind, including a personal observation, PASCHEFF (*Archiv. d'ophtal.*, October, 1910, p. 634) finds that when an aneurysm causes paralysis of the third nerve alone, it is uniformly seated upon the trunk of the internal carotid, between the origins of the anterior and posterior communicating arteries. When the aneurysm involves the origin of the posterior cerebral artery, the paralysis of the third nerve is accompanied by paralysis of the corresponding facial. The only subjective symptoms (besides the diplopia) are pains in the head and constant noises upon the same side as the aneurysm. These cases always end fatally, usually quite suddenly, shortly after the appearance of the paralysis.

Detachment of the Retina Treated by Tuberculin.—Poorly or non-vascularized tissues like the tendons, aponeuroses, serous membranes, cartilage, being the seats of election of bacillary lesions, DOR (*Clin. ophtal.*, August, 10, 1910, p. 362) raises the question whether the vitreous body might not be added to the same list. Detachment of the retina, being secondary to retraction of the vitreous, presupposes antecedent disease of that body. The fact that the lesion is particularly apt to occur in myopia argues that the latter simply creates the anatomical conditions favorable to such an occurrence, just as osseous tuberculosis affects by preference bones which have undergone too rapid growth. Upon systematically questioning patients suffering from detachment, he has frequently found an antecedent pleurisy, arthritis, adenopathy, chronic rheumatism, or chronic bronchitis. Based upon such considerations, he has treated five cases of detachment by tuberculin, with complete and permanent re-application of the retina in three cases, re-application followed by relapse in one, and failure in a fifth—a degree of success which can certainly not be the result of coincidence.

The Principal Meridians in Astigmatism in Relation to the Lateral Inclination of the Head.—ORESTE (*Ann. d'ocul.*, June, 1910, p. 459) has studied the changes in the direction of the principal meridians in astigmatism when the head is inclined laterally. He finds that in the majority of cases the eye executes a movement of rotation about its antero-posterior axis in the inverse direction, to compensate, in part, the lateral inclination of the head. This movement of compensatory rotation, nil in some cases, varies in general between one and four to five degrees on the average, but may attain as much as eight. In some cases such compensation takes place in a single direction only. It has been supposed that when a vertical line is fixed, and the head is thence turned laterally, the line continues to be seen in its vertical position because the eye tends to return to the primitive position. The compensation possible, however, is too weak to overcome inclination of the head of any considerable

amount. Hence the correct vision of vertical lines under these circumstances is a phenomenon of psychic origin due to the fact that the subject is conscious of the position of his eyes, and makes the requisite allowances.

Unilateral Cataract from Electricity.—BICHELONNE (*Annal. d'oculist.*, August, 1910, p. 108) concludes that, considering the number of instances of accidental contact with currents of high power, cataract from such a cause is rare, a circumstance which may be due to the fact that the crystalline lens becomes affected only when one of the points of contact is not far from the eye itself, which is also a rare occurrence, for the contact is most commonly made by the two hands. For the same reason the cataract is almost always localized upon the affected side. Voltage seems to have little influence. The opacity of the lens is tardy in appearing, and seems to be dependent not upon lesions of the nerve centres or peripheral nerves, but upon a local trophic disturbance, circulatory or otherwise, which manifests itself upon an organ more vulnerable than the neighboring parts.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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The Interrelation of Glands of Internal Secretion. III. Chemotaxis.—BERTELLI, FALTA, and SCHWEEGER (*Ztschr. f. klin. Med.*, 1910, lxxi, 23) have studied the effect which a number of substances show upon the cells of the circulating blood when injected subcutaneously into dogs. These substances were adrenalin, pilocarpine, atropine, sodium nitrite, extracts of the infundibular and glandular portions of the pituitary body, cholein hydrochlorate, and ergotin. They find that, in general, these substances may be divided into two groups according to their effects upon the blood cells, or rather upon the blood-forming organs. The first group, represented by adrenalin and atropine, produces a rapid, marked, and prolonged hyperleukocytosis, accompanied at first by increase in the eosinophilic cells and later by disappearance of these cells from the blood. Adrenalin, moreover, gives rise to a rapid polycythemia, for the red cells increased ten minutes after injection 40 per cent. and after five hours at times 70 to 100 per cent. The second group includes all the other substances, and their action is characterized by the following effects: At first there is transitory accumulation of the neutrophilic leukocytes in the internal organs, and especially in the liver, while at the same time there is an actual increase in the mononuclear cells in the peripheral circulation and more or less pronounced hypereosinophilia. Later, the blood shows an increase in the neutrophilic and eosinophilic leukocytes. The action of adrenalin and of the substances in the second group is not believed to be dependent upon

blood pressure changes nor upon changes in the caliber of the capillaries, nor does the explanation based by Ehrlich upon the theory of negative and positive chemotaxis seem adequate to explain the action of these substances. The authors point out the fact that the substances in each of the two groups have in common a similar action on the nervous system: adrenalin, for instance, acting as an excitant to sympathetic nerves. This analogy is suggestive in connection with the prompt response of the cells of the blood to injections of adrenalin, and there is good reason to suppose that the substances in the first group act as stimulators of the bone marrow and of increased blood formation. The explanation for local chemotaxis is not clear, but it seems at least that when substances arrive in the circulation the chemotactic stimulus runs parallel with the variation in tone of the sympathetic nervous system. The authors suggest that the variations in the output of cells from the blood-forming organs may be in part controlled by internal secretions.

The Venous Pulse in Fibrillation of the Auricle in the Mammalian Heart, with Reference to the Venous Pulse in Pulsus Irregularis Perpetuus.—Rihl (*Ztschr. f. exp. Path. u. Therap.*, 1910, vii, 693) has studied the graphic tracings made by means of a special tambour from the jugular veins in dogs in which fibrillation of the auricle was produced by electrical stimulation of the vagus nerve. The carotid and subclavian arteries on the side from which the venous tracings were made were tied off to prevent a transmitted pulsation to the vein. The activity of the auricle and ventrical was recorded by the suspension method. Since the method of tracing the waves in the jugular vein was the same as that employed in man, the venous tracings were comparable to those taken from human subjects. Before the production of fibrillation of the auricle the venous pulse showed a well-marked *a* wave, followed by the two waves of ventricular origin. After the establishment of fibrillation of the auricle the *a* wave disappeared entirely from the venous tracings, leaving only the two waves of ventricular origin. The tracings therefore corresponded exactly with those which are obtained in man in pulsus irregularis perpetuus, not only in the disappearance in the wave produced by the contraction of the auricle, but even in certain minute details concerning the size and variation in the waves dependent upon the ventricular action.

The Keith-Flack Node in the Production of Cardiac Arrhythmia.—The recent investigations of Walter Koch upon the anatomy and pathology of the bundle of muscle described originally by Keith and Flack as lying between the vena cava superior and the auricle, has suggested that certain pathological changes in this region may possibly play a part in the production of some of the cardiac diseases which at the present are not perfectly understood. Thorel, too, has described a bundle of muscle fibers which connects the sinus node with the node of Tawara in the auricle. Owing to the probable interest which the Keith-Flack node may have for clinical medicine, MacKenzie already having suggested that lesions of this muscle bundle may be connected with perpetual arrhythmia, Jager (*Deut. Arch. f. klin. Med.*, 1910, c, 1) has destroyed the node in 15 dogs and 45 cats in order to see what effect

this might have upon the rhythm of the heart. The node was destroyed by a hot instrument, serial sections being made later to determine whether the entire muscle bundle was destroyed. The experiments were successful in 8 cats and 6 dogs, but total destruction of the Keith-Flack node failed to give rise to any arrhythmia of the heart. Jäger therefore concludes that the results obtained by Hering after cutting the venoauricular junction, namely, cessation of the beat of the auricle, was not due to injury to the node, and that such injuries do not give rise in dogs and cats at least to a disturbance in rhythm of the heart.

The Substitution of Strontium for Calcium in Bones.—Lehnerdt (*Ziegler's Beiträge*, 1909, xlvi, 468; 1910, xlvii, 213) in a series of interesting experiments has fed pregnant rabbits strontium phosphate. Usually calcium was eliminated from the diet, but in a few experiments strontium phosphate was simply added to the animal's ordinary food He has found that by this means profound changes are brought about in the bones of the young of the pregnant rabbits. These consist in deformities, principally of the long bones, and resemble to a certain extent the pathological lesions of rickets. They are, however, not exactly the same, and should be termed pseudorickets. The action of strontium is that of a formative stimulus to the bones, and the absorption of large quantities of this salt by the fœtus in the absence of calcium gives rise to spongy osteoid formations at the ends of the bones, with rapid union and great decrease in the absorption of bone. When calcium is fed in small amounts together with strontium, the spongy outgrowths show some calcification, but with an increase in the amount of calcium the abnormal changes in the bone diminish. In a second communication Lehnerdt describes some further experiments carried out under the same conditions with newborn puppies. The results here were much the same as in the first series of experiments, and the changes occurring in the bones were those of a pseudorickets, consisting in diminished resorption of bone with exaggerated apposition in bones which were of normal length. When fractures occurred they healed with the formation of an excess of callous; when calcium was added to the food the spongy osteoid growths became calcified. The action of strontium appears in many ways like that of phosphorus, though there are certain differences in the effects of the two drugs. Lehnerdt finally suggests that strontium may be of therapeutic value in osteomalacia, in such conditions as osteoporosis when there is an increased resorption of bone, and in conditions in which callous formation is delayed and slight.

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